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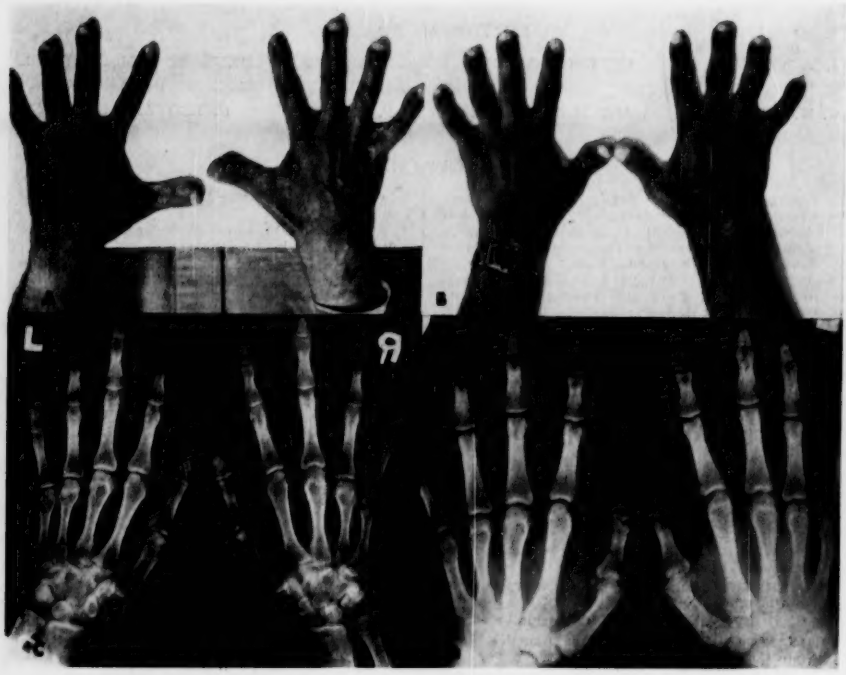

TWO BRACHYDACTYLOUS TRAITS IN ONE FAMILY

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Mormoniella Merry-Go-Rounds

Mitotic Instability in the Blackberry

Recessive Susceptibility to Poliomyelitis

Chromosomes and Sex in the Blood Fluke

VOLUME 48 JANUARY-FEBRUARY, 1957 NUMBER 1

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METAPHASE CHROMOSOMES OF *S. DOUTHITTI*

Figure 1

A—*F* shows metaphase chromosomes in cercarial embryos of *Schistosomatium douthitti* and were drawn to the same scale with the aid of a camera lucida. *A*—Male diploid, from section; *B* and *C*—female diploid, from sections; *D*—male diploid, from squash; *E*—female diploid, from squash; *F*—male triploid, from section.

CHROMOSOMES AND SEX IN SCHISTOSOMATIUM DOUTHITTI

Trematoda: Schistosomatidae

ROBERT B. SHORT*

ASIDE from their medical importance, trematodes of the family Schistosomatidae have attracted attention because they are dioecious. This condition, accompanied by marked sexual dimorphism in adults but not in larval stages, has stimulated interest in sex determination and differentiation in this group.

Cort[3] proposed the hypothesis that sex in schistosomes is determined in the fertilized egg and all cercariae resulting from a single miracidium are of the same sex. Evidence for this was the finding that usually cercariae of only one sex emerged from single naturally infected snails. In more recent years this hypothesis has been repeatedly supported by results of experimental infection of snails with *Schistosoma mansoni, S. japonicum* and *Schistosomatium douthitti*, so that today it is commonly accepted that an infection produced by a single miracidium will result in cercariae of only one sex. In view of these findings it seems reasonable to assume that sex in schistosomes is already determined in the miracidium stage, thus in the zygote from which the miracidium develops, and that sex determination might be explainable on a chromosomal basis.

Only a few reports on chromosomes of schistosomes occur and the results are conflicting. Earlier papers on *Schistosoma haematobium*[9] and *S. japonicum*[5, 12] reported sex chromosomes with the male heterogametic. However, more recent cytological studies on *S. japonicum*[7] and *S. mansoni*[11] have failed to reveal heteromorphic chromosomes in either sex.

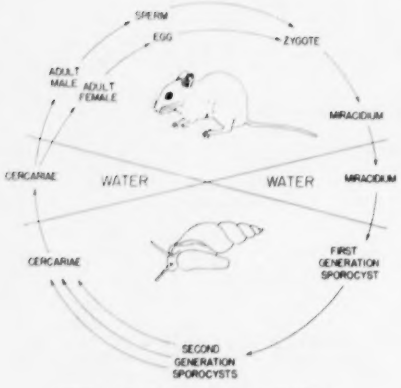
The present paper is concerned with chromosome number and morphology in *Schistosomatium douthitti* (Cort.). A heteromorphic pair of chromosomes has been identified and the evidence indicates that the female is heterogametic. An abstract of these results has been published.[13]

Materials and Methods

Schistosomatium douthitti is a blood fluke which occurs in nature as adults in muskrats and field mice and as larvae in lymnaeid snails. Natural infections have been reported from Illinois, Massachusetts, Minnesota and Wisconsin. The stock of *S. douthitti* used in the present work was kindly supplied by Dr. I. G. Kagan from near Chicago, Illinois.

The fluke's life cycle is illustrated in Figure 2 and is typical for schistosomes. *S. douthitti* can be maintained in the laboratory through many generations (for methods see Kagan, Short and Nez[8]). Laboratory mice and hamsters are commonly used as definitive hosts. The adult male and female flukes live within the portal system; here the females deposit eggs, many of which pass through the intestinal wall and are voided with the feces. Upon reaching water, miracidia hatch from the eggs. These larvae penetrate snails and each miracidium metamorphoses into a first generation sporocyst. Within each first generation sporocyst many second gen-

* Department of Biological Sciences, Florida State University, Tallahassee. This investigation was supported by research grant E-346 from the National Microbiological Institute of the National Institutes of Health, Public Health Service. The author wishes to express his gratitude to Dr. Margaret Y. Menzel for her aid which included making many of the chromosome analyses and the drawings for Figure 1*D-F*. My appreciation is also expressed to Dr. Martha M. Nez and Miss Mary Ann Joyner for their competent technical assistance.



THE FLUKE'S LIFE CYCLE

Figure 2

This diagram illustrates the life cycle of *Schistosomatium douthitti*, and is typical for schistosomes.

eration sporocysts develop; these migrate to the snail's digestive gland and each produces many forked-tailed cercariae. The cercariae emerge from the snail host, swim about in the water, and penetrate a mammal if present.

Asexual reproduction in the sporocyst generations makes possible the development of thousands of cercariae from a single miracidium. As mentioned above, evidence indicates that the miracidia are sex determined, although morphological differences are not evident. Thus, a snail infection produced by a single miracidium (representing one zygote) furnishes for study a great number of developing cercarial embryos, all of the same sex.

During the present investigation chromosomes were studied in male and female cercarial embryos and a few solitary germinal cells from unisexual snail infections of known sex. Snail infections were obtained by exposing young laboratory-reared specimens of *Lymnaea palustris* to single miracidia from bisexual infections in mice. The sex of cercariae from each infection was determined by mouse exposure. Snails digestive glands containing sporocysts with cercarial embryos were fixed in Bouin's, Carnoy's and Sanfelice's solutions after removal of the snails' shells. Material fixed in all three solutions was sectioned at 8 and 10 microns and stained with iron hematoxylin. Some sectioned material was also stained with Feulgen's stain after fixation in Sanfelice. All these techniques gave satisfactory results. Aceto-carmine squashes were also made of material fixed in Carnoy 3:1 from about one-half the snail infections. This method gave excellent chromosome pictures and proved especially helpful for rapid analysis.

Observations

Metaphase and late prophase chromosomes were studied in 25 male and 25 female infections. The diploid number in each sex is 14, confirming Nez's[10] counts in her study of gametogenesis. In sectioned material metaphase chromosomes often were observed with their arms as well as their centromeres almost entirely in one plane. This behavior rendered chromosomes in sectioned material from snails more favorable for analysis of morphology than those in adult worms.

The individual chromosomes could all be identified in many cells in both sections and squashes and have been labelled in the figures. In 24 of the male infections the chromosome complement (Figure 1*A* and *D*) was: one large V-shaped pair (z); three medium-sized V-shaped pairs of different sizes (a, b, c); two medium-sized J-shaped pairs (d, e), one with satellites (e); and one small pair with median or submedian centromeres. In 24 of the female infections the complement (Figure 1 *B*, *C* and *E*) was the same as that of the males, except that instead of the two large V-shaped chromosomes there was one large V plus a medium-sized rod-shaped element (w). Since the 24 males all had two large V-shaped chromosomes, and the 24 females all had only one large V, plus one rod not present in the male, it is inferred that these are sex chromosomes (designated *Z* and *W* respectively) and that the female is the heterogametic sex.

At metaphase the *Z* chromosomes in sectioned material were about four microns long and about twice this length in squashes. In some preparations a secondary constriction was evident in one arm of the *Z* (Figure 1*B*), and in a few instances a region of one arm appeared negatively heteropycnotic (Figure 1*C*).

The above results were obtained from analysis of metaphase and late prophase chromosomes in 198 cells from the male infections and 238 cells from female infections. The evidence leaves little doubt that the

diploid male genotype is *ZZAA* and the female genotype *ZWAA*.

In two additional snail infections, one determined to be male, the other female, more than 14 chromosomes were consistently counted. Analysis of five plates from the male material indicated that the cells were triploid (Figure 1*F*), and of the constitution *ZZWAAA*. In material from the female infection, more than 14 chromosomes were also evident, and in seven plates 21 chromosomes were counted with only one *Z* present. Although no cells were found which were sufficiently clear to allow identification of all the chromosomes, the evidence suggests that the female infection was also triploid, with the constitution *ZWWAA*.

Discussion

The evidence seems clear that the female *S. douthitti* is heterogametic for sex despite the lack of direct observation of ova with two different chromosomal constitutions. Heterogamety of the female stands in contrast to male heterogamety as reported in *S. haemotobium* by Lindner[9] and in *S. japonicum* by Faust and Meleney[5] and Severinghaus[12]. The presence of a heteromorphic pair of chromosomes also differs from the findings of Ikeda and Makino[7] in *S. japonicum* and Niyamasena[11] in *S. mansoni*. According to Lindner's results on *S. haematobium* the male has 14 chromosomes (2*X*, 12*A*) and the female presumably 16 (4*X*, 12*A*). Faust and Meleney reported similar findings in *S. japonicum* except that the male was considered to have 15 chromosomes (*X*, 14*A*). Severinghaus later disagreed with the findings of Faust and Meleney and stated that in *S. japonicum* the situation was the same as Lindner had reported for *S. haematobium*.

The results of these early studies were based primarily on spermatogenesis and are not in agreement with the more recent work of Ikeda and Makino[7] on *S. japonicum* and Niyamasena[11] on *S. mansoni*. In both species these authors found 16 chromosomes (diploid) with no evidence for a heteromorphic pair in either sex. Unpublished preliminary results in the author's laboratory confirm this finding in *S. mansoni*. Thus, it appears that members of the genus *Schistosoma* possess 16 chromosomes in contrast to 14 in *Schistosomatium* and, in *S. japonicum* and *S. mansoni* at least, there are no morphologically distinguishable sex chromosomes.

The chromosome morphology of *S. douthitti* also differs from that of *S. japonicum* as shown by Ikeda and Makino[7]. In *S. japonicum* the diploid complement of both sexes is: two relatively large V-shaped pairs, one with median, the other with submedian centromeres; and six pairs of rod-shaped ones gradually diminishing in length. Thus in *S. japonicum* most of the chromosomes are rod-shaped while in *S. douthitti* the only rod-shaped chromosome appears to be the *W*. It is hoped that studies on additional species of schistosomes will enable such comparisons to throw light on the evolutionary history of the group.

While it seems evident that *S. douthitti* possesses morphologically differentiated sex chromosomes, and that the female is heterogametic, the exact genetic nature of the sex determining mechanism cannot be deduced from the present work. However, the chromosome constitution of the triploid male infection (*ZZW*) suggests that the presence of a single *W* does not make the individuals female as it apparently does in the axolotl (which also has female heterogamety) in *ZZW* and *ZZZW* genotypes[4,6]. The evidence leaves open the possibility that sex determination in *S. douthitti* is in accord with the genic balance theory which Bridges[1,2] formulated from results on *Drosophila melanogaster* (in which, however, the male is heterogametic). If the situation in *S. douthitti* were strictly analogous to that in *Drosophila* (except with the sexes reversed) the net effect of the *Z* chromosome would be male-determining, while the effect of the autosomes would be female-determining, with the effect of one *Z* outweighing that of one set of autosomes. However, the data do not exclude the possibility that the female-determining genes of *S. douthitti* are largely in the *W* chromosome. In either case if the balance effect were analogous to that in *Drosophila* the triploid male *S. douthitti* cercariae (*ZZWAAA*) should have developed into inter-sexes and the triploid females (*ZWWAAA*) into "super-females." Unfortunately the worms which resulted from these two triploid infections were discarded (prior to chromosome studies) after determination of sex, so that any sexual abnormalities not evident in living specimens examined with a stereobinocular microscope would have escaped notice. The fact that the worms were routinely classified as to sex indicates that they had at least the external form characteristic of the male in one instance and the female in the other.

These two triploid examples were discovered among the first 28 snail infections examined and suggested that natural triploids may occur rather frequently. With the hope of finding additional triploids, material from another series of 22 snail infections was studied. All these proved to be normal diploids.

Summary

Chromosomes of *S. douthitti* were studied in cercarial embryos of known sex. The diploid chromosome number for both sexes is 14. All of the chromosomes can be identified morphologically in favorable cells. The female possesses a heteromorphic pair of chromosomes and is interpreted as being heterogametic (*ZW*). Two triploid infections were

found, one male (*ZZW*), the other female (*ZWW*). The fact that individuals with the chromosome constitution *ZZW* were males suggests that the *W* chromosome is not strongly female-determining.

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ON THE ENDOSPERM NUCLEUS OF ALLIUM

YUKIO KATO*

AMONG the cytological studies on endosperm nucleus, we are particularly interested in the chromosome aberrations in connection with both the mosaic formation in maize (Clark and Copeland[4]) and with the failure of interspecific hybridization (Kihara and Nishiyama[8], Cooper and Brink[5], Brock[2]), and in the enlargement of nuclear size associated with polyteny (Duncan and Ross[6]) and polyploidy (Punnett[12]).

Duncan and Ross[6] reported nuclear change and nuclear activity in developing maize endosperm. They showed that this nuclear change was due to an endomitotic process in which there was an increase in the number of strands per chromosome as well as an increase in breadth and length of the individual strands. More recently, Punnett[12] reported an endomitotic cycle resulting in hexaploid cells in maize endosperm eight days old.

The role of the endosperm in seed development has been reviewed by Brink and Cooper[1]. They considered that the failure of the fertilized ovule to develop into a mature seed was the result of an abnormal development in the endosperm.

The present study is to elucidate the following points: 1) the endomitotic cycle, 2) the highly polyploid division, and 3) abnormal mitosis in the endosperm tissue of the selfed Welsh onion.

Materials and Methods

At 3, 6, 14 and 21 days after selfing, immature seeds of *Allium fistulosum* were dissected under the binocular microscope into embryo, endosperm and seed coat by manipulation with a sharp needle. Thus isolated these endosperm tissues were fixed in absolute alcohol-glacial acetic acid (3:1) and then squashed in aceto-orcein. In order to see the mutual position within endosperm tissue, an entire immature seed was embedded, sectioned, and stained with thionine or tholuidine blue. In the Welsh onion, cytological events could be more readily followed by squashing than in sections. Three localized areas in the endosperm were selected for study: 1) the adgerminal part lying between the embryo and the adjacent enveloping tissue, 2) the peripheral part on the abgerminal side and 3) the central part of the endosperm.

Observations

Highly polyploid nuclei. The materials from three to six days after anthesis could not be sufficiently examined because they were too small. In the 14-day-old material, the mitosis of the cells in the peripheral part of the endosperm is quite normal, and 24 chromosomes (triploid number) were counted at metaphase. In addition to the normal mitoses, each of the 24 chromosomes united at the single kinetochore at prometaphase. Then, all the stages of an endomitotic cycle could be observed.

The nuclei of the cells in the peripheral part do not appear to develop further. The nuclei in the central part, on the other hand, increase up to 500 times or more their original volume. They grow extensively but lose their mitotic activity rapidly. Therefore, nuclei in the central part of the endosperm are quite different in behavior from those in the peripheral part. The nuclei which increased 500 times seldom appeared in the nuclear division. Roughly 800 chromosomes (approximately 100-ploid) could be counted in such cells. An exact count of the number of chromosomes was often impossible. Such highly polyploid cells found in the endosperm may result from repetition of the following phenomena: 1) repeated duplication of chromonemata without subsequent

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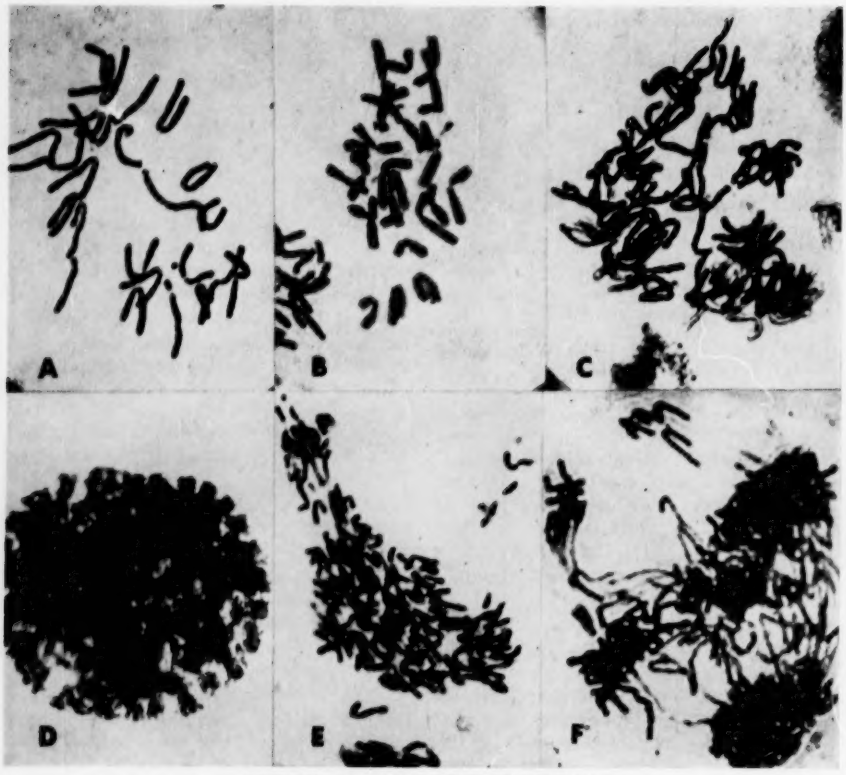

MITOSIS OF ENDOSPERM NUCLEI

Figure 3

Some aspects of mitosis, endomitosis and cytological abnormalities in Welsh onion endosperm. Fixation in acetic alcohol, stained with aceto-orcein. *A*—Metaphase showing 24 chromosomes (triploid number). *B*—Metaphase showing tetrachromosomes. *C*—48 chromosomes resulting from 24 tetrachromosomes. *D*—Multi-stranded chromosomes. *E*—Irregular separation in anaphase. Near-10-ploid. *F*—Anaphase showing irregular separation and sticky bridges.

mitosis, resulting in polytene chromosomes, 2) duplication of chromosomes during the resting stage and 3) failure of anaphase separation followed by restitution. In fact, the increase in the number of strands making up each chromosome was frequently observed in the nuclei of the central part. The chromosome appears rope-like being made up of many fine strands.

Chromosome breakage and reunion. As mentioned above, the nuclear division of the cell in the peripheral part of the endosperm proceeded normally, while in the central part various patterns of abnormal mitoses were often found. Figure 3*E* and *F* represents respectively the disorientation of approximately 80 chromosomes and the sticky bridges at anaphase. These cells did not exhibit any fragmentation. In addition to sticky bridges, the real chromatin bridge, probably derived from chromosome breakage and reunion, was abundantly observed. Several anaphase bridges obviously prevented the separation of the chromosome groups. The telophase followed by the irregular anaphases also naturally shows many abnormal features.

CYTOLOGICAL ABNORMALITIES IN THE ENDOSPERM NUCLEI

Figure 4

Endosperm nuclei of selfed Welsh onion. *A*—Metaphase of a highly polyploid nucleus showing acentric fragment. *B*—Metaphase showing rings and acentric fragments. Aceto-orcein squash.

Chromosome breakage seems to be associated with the polyploid condition. The frequency of aberrant cells was examined in relation to the polyploid level (Table I).

Chromosome aberrations appeared only in highly polyploid cells, i.e. metaphase and anaphase showing structural change of chromosomes are in highly polyploid cells (Figure 4*A* and *B*). In some cells the breakage was too frequent to be scored. The breakage produced ring-shaped, dicentric, minute and rod-shaped fragments. The minute fragment is a type of structural change occurring frequently. As a rule, minute chromosomes are rarely observed in *Allium* root-tip cells. The formation of tripolar spindles is seldom seen in highly polyploid cells, but spindle abnormalities are not necessarily associated with chromosome breakage (Kato[7]).

Discussion

Polyploidy and mitotic abnormalities in the endosperm observed by the author resemble closely those in differentiated root tissue. The nuclei of differentiated cells differ from those of the meristematic cells. Levan[10] and Therman[12] stated that the high degree of polyploidy in differentiated tissues of plants may be an indication that the cells are degenerating. In such degenerating cells, the functional spindle is not formed nor does normal chromosome reproduction occur. This is, then, a pathological form of polyploidy. A similar consideration can be applied to the present observation. The nuclei of endosperm tissue are evanescent and soon degenerate. In this respect the endosperm is just like the tapetum which in many plants shows the polyploid nature and mitotic abnormalities. Generally speaking, spontaneous chromosome aberrations occur under conditions belonging to one of the three following types: 1) extremely unfavorable environmental conditions, 2) special genotypic conditions, and 3) differentiation within an organism. Our studies fall into the last category. Levan and Lofty[9] explained the structural change of chromosomes in differentiated cells as due to chromosome decay in aging tissues. It seems evident that degenerating changes may, in certain circumstances, take place in the chromosomes. The term "degeneration" in this connection implies a process leading to the death of the structure concerned. It is well-known that indole-3-acetic acid compels a division of the resting nuclei showing various patterns of polyploidy and also acts as a detector of structural changes of chromosomes in the differentiated cells. Luckwill[11] and others have pointed out that plant hormones settle abundantly in the endosperm. In other words, the site of production of plant hormones is a layer of nutritive tissue. In addition, it may be assumed that the "Necrohormone" in Haberlandt's signification is produced from the degenerating endosperm. These agencies may cause the giant nuclei to divide.

Chromosome breakage is significant for the failure of interspecific hybridization or for genetic varigation. The accumulation of abnormalities reduces the mitotic activity and the failure of endosperm formation causes the failure of development in the seed. It is to be noted that abnormalities of the endosperm development occur especially in the part adjacent to the embryo or in the antipodal cells. Therefore, the abnormalities observed in the pres-

TABLE I. Frequency of aberrant cells with bridges and/or fragments in the endosperm *

Polyploid level	Number of total cells examined	Number of cells with bridges	Number of cells with fragments‡	Number of cells with bridges and fragments	Percentage of aberrant cells
Near—3*n*	58	0	0	0	0
Near—6*n*†	33	0	0	0	0
Highly polyploid nuclei	38	7	12	16	92.1

*The abnormalities were analyzed in cells at metaphase and anaphase.
†The cells having paired chromosomes in metaphase were regarded as near-6*n* in this datum.
‡Fragments included ring-shaped, dicentric, minute and acentric ones.

ent study must be distinguished from those found in interspecific hybridization. Cave and Brown[3] reported that the cytological abnormalities in ovules of *Lilium formosanum* fertilized by irradiated sperm nuclei at a X-ray dosage of 4000r are usually represented by large, irregularly lobed interphase nuclei and micronuclei. Some nuclei were polyploid and others probably polytene. During the course of our studies to overcome the self-incompatibility in the lily, the present author found many abnormal nuclei in the endosperm of *Lilium longiflorum* treated by growth-regulators and also of self-pollinated *L. formosanum*. The typical endosperm nuclei in the peripheral part are relatively constant in size, while nuclei in the central part have increased, being three or four times, often nearly 10 times, as large as those of the peripheral part. The most characteristic feature observed in the central part of the endosperm is the giant, lobed nuclei, which are often irregular in shape. Micronuclei were found in some cases. All giant nuclei become necrotic and degenerate at a later stage. The features of these abnormal nuclei are similar to those of Cave and Brown's figures. They suggested that mechanical difficulties, genetic unbalance and toxic effects of irradiation can not be eliminated as a cause of cytological abnormalities in the endosperm, but the present author considers that further study is needed to determine the factors which induce these abnormalities. It seems probable that the differentiation within the organism plays an important role in the patterns of cytological abnormalities. In the present material, pollen mother cells revealed the regular meiosis, and thus pollen grains were plentiful. Furthermore, no embryos showed cytological abnormalities.

A very high polyploidy and polyteny has been found in some insect tissues, but the polyploidy found in plant cells is restricted to relatively small multiples. This may be due either to self-regulating reduction or to lethal effects.

Summary

Nuclear divisions in the central part of *Allium* endosperms are characterized by endomitosis, high polyploidy and cytological abnormalities. These phenomena are concerned with the differentiation within the organism and can be regarded as an indication that the cells are degenerating.

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MITOTIC INSTABILITY IN RUBUS

DONALD M. BRITTON AND J. W. HULL*

AN earlier paper reported the finding of widely differing chromosome numbers in individual plants in the progenies of Boysen and of Young blackberries crossed with colchicine-treated Eldorado[1]. Two characteristics of these plants implied the action of some mechanism resulting in mitotic instability: 1) the absence of appreciable numbers of chromosome number determinations which could have been derived through polysomaty; and 2) the finding of complete and wide ranges of aneuploid chromosome numbers in single individuals.

Subsequently, additional unstable plants of *Rubus* have been found in the progenies of many different crosses. These plants and their asexually propagated clones have been observed and studied cytologically to determine the mechanisms causing mitotic instability.

Materials and Methods

All the unstable *Rubus* plants discussed in this paper were grown from seeds obtained by carefully controlled crosses made in the greenhouse. Leaf-bud cuttings of the mitotically unstable plants were propagated in coarse sand under constant mist in an outdoor propagating frame. Root-cuttings were made in the usual manner of commercial practice. All seedlings and clones were grown in individual pots in the greenhouse.

The cytological methods for root-tip determinations were the same as used by Britton and Hull[1]. Also, the axillary leaf-bud squashes were made in the same manner. In addition, many methods were tried in order to show the mitotic spindles clearly as well as the chromosomes. While none were completely satisfactory, the best results were obtained by fixing root-tips in CRAF for 35 minutes, rinsing in running water for 5-10 minutes, and storing in 70 percent ethyl alcohol until such time as the root-tips were required. The root-tips were then hydrolized for 20 minutes in normal hydrochloric acid at 60° C., stained in leuco-basic fuchsin (Feulgen) for two hours, and squashed in iron-aceto-carmine.

Observations

Crosses which have produced one or more mitotically unstable plants among their respective progenies are presented in Table I. It has not always been possible to recognize such unstable individual plants on the basis of their phenotypes. However, many have a pronounced "checking" on their leaves (Figures 5*B* and 6). These "checks" or mosaic areas are of a lighter green color and are thinner than the rest of the leaf, but rarely involve as much as an entire leaflet. While all plants exhibiting this mosaicism have been found to have various chromosome numbers in their roots, a few mitotically unstable plants have been found with no evidence of "checking" in their leaves.

In one population of 66 seedlings of Eldorado (4*x*), ($2n=28$) selfed, five of the young plants were determined to be $2n=42$ by means of axillary leaf-bud squashes. These 6*x* plants presumably resulted from fertilization of unreduced eggs by reduced pollen grains—an event not uncommon in *Rubus*[2]. In contrast to the regular-appearing leaves of 4*x* Eldorado selfed seedlings (Figure 5*A*), the 6*x* seedlings exhibited, in addition to their symptoms of higher ploidy[5] a noticeable degree of "checking' or mosaicism in their leaves (Figure 5*B*). Cytological examination of their root-tips indicated that many cells had 42 chromosomes and others had fewer—

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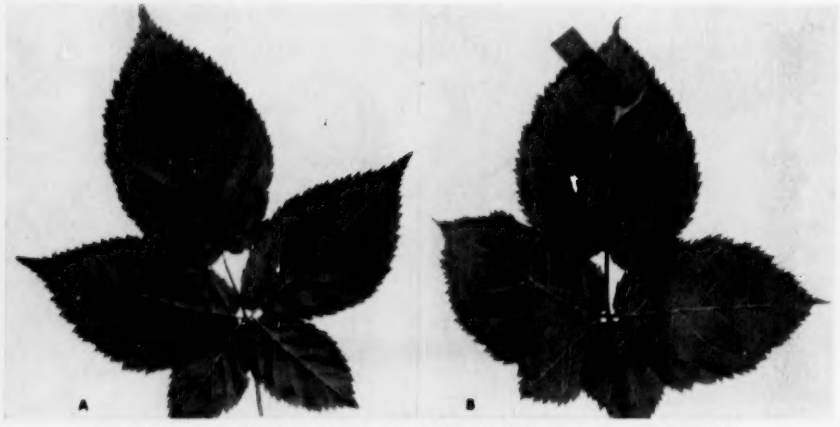


LEAVES OF SEEDLINGS OF SELFED ELDORADO BLACKBERRY

Figure 5

A—Leaf of seedling (4*x*) of Eldorado selfed to compare with *B*. *B*—Leaf of seedling (6*x*) of selfed Eldorado. Note the change in shape, serration, angle of leaf veins, and puckering. The arrow points toward a thinner, lighter area of mosaicism. Two other such areas are present in the lower right leaflet.

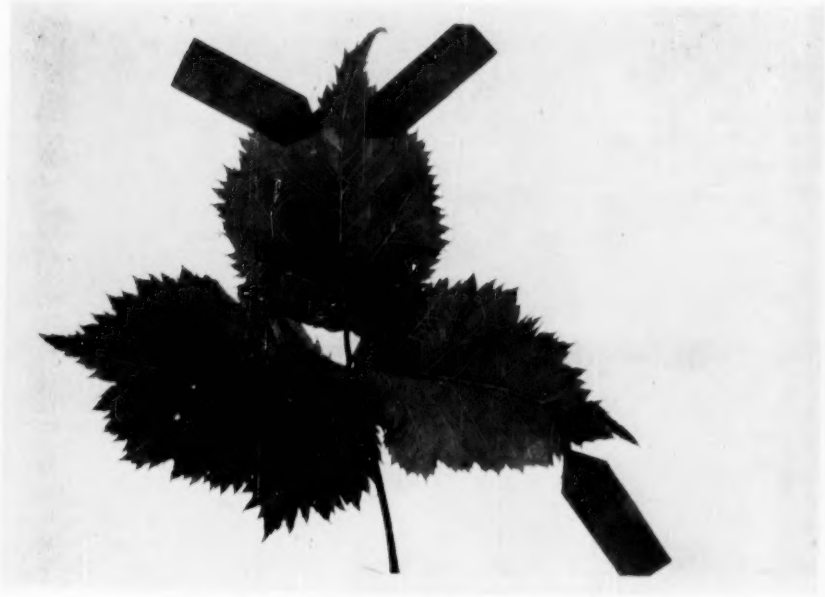
some markedly fewer (Table II). No evidence of mitotic instability has been found in 4*x* Eldorado plants or 4*x* selfed seedlings of Eldorado.

As in the case of the 6*x* seedlings of selfed Eldorado, most unstable plants of other *Rubus* seedling populations have not been notably weak, dwarf, or otherwise abnormal in appearance. To the contrary, many were among the most vigorous of their respective populations. However, a few dwarfs have been found with unexpectedly high chromosome numbers which appeared most frequently to have resulted from fertilization of unreduced eggs. Many of these produced root suckers with lower chromosome numbers which had markedly increased vigor.

Leaf-bud cuttings of the 28 original unstable plants all grew into plants having first season shoots which appeared to be identical phenotypically to those of the corresponding original mother plants. Chromosome number determinations made from root-tips of these clones indicated that they were still mitotically unstable.

Leaf-bud cuttings were made from these plants that had been grown from leaf-bud cuttings, and again the phenotypic characteristics of the original shoot of the mother plant were maintained. However, cytological studies of their root-tips showed that the mitotic instability was still present. Chromosome number determinations from root-tips made when these latter plants were only a few weeks old revealed many high numbers as well as lower numbers. Later in the season, it was often possible to find only low chromosome numbers (e.g. 20-35). At this later time, axillary leaf-bud squashes were made which showed many cells with the high chromosome number of the original shoot of the mother plant.

On the other hand, root-cuttings from single unstable plants have produced plants exhibiting a wide range of phenotypes quite unlike the mother plant or its parents. In the case of one of the plants previously reported (Plant No. 1, Table III, Britton and Hull[1]), 27 plants were produced from root-cuttings. Two were weak and died shortly after sprouting and the remaining 25 could be classified into four groups of quite different



MOSAIC LEAF OF A MITOTICALLY UNSTABLE PLANT

Figure 6

Leaf from a mitotically unstable hybrid (Boysen × (8x) Eldorado). Arrows point to some of the more striking areas of mosaicism. The whole area of the leaflet below the midrib and to the left of the arrow at lower right, is of similar appearance to the smaller areas of "checking" on other leaflets.

phenotypes with minor variations within each group. None were at all similar to the phenotype of the original shoot of the mother plant.

An example of three canes from the roots of another unstable plant is shown in Figure 7. The leaves on primocane *B* are thick, irregularly toothed, puckered, and divided into 3-5 leaflets haphazardly. Those on primocane *A* are extremely uniform in appearance. All are thin, regularly toothed, smooth, and divided neatly into three leaflets. Primocane *C* has leaves which are markedly "cutleaf" or incised, in a way unlike the mother plant or either of its parents. Further, the cane is more spindly, wiry, and weak than that of either *A* or *B*.

Twenty leaf-bud cuttings were made from primocanes *A*, *B*, and *C* (Figure 7), and the plants which grew from these leaf-bud cuttings had the phenotypes of the canes from which they were propagated. Cytological studies of root-tip squashes of the five plants from these leaf-bud cuttings of primocane *B* showed that in each plant many cells had 56 chromosomes, but that there were other lower chromosome numbers present. Root-tip squashes for the eight plants from primocane *A* showed that all cells had 31 chromosomes, and root-tips of the seven plants from primocane *C* had cells with 29 chromosomes only. Thus, propagation of plants with the high and unstable chromosome number of primocane *B* resulted in plants which were still mitotically unstable, whereas propagation from primocanes *A* and *C* of lower chromosome number, resulted in plants which were mitotically stable.

A few isolated cells have been seen in root-tips of some unstable plants with as few as 10 or 11 chromosomes—below

the gametic number of either parent. However, when propagations of root-cuttings or root suckers have been made of the plants in which these low chromosomt numbers were observed, the plant with the lowest chromosome number obtained had 26 chromosomes.

Cytological Observations and Discussion

Cytological studies of root-tip squashes from the 6*x* seedlings of selfed Eldorado showed many cells with fewer than 42 chromosomes (Table II). In addition there were seen configurations which suggested how these lower chromosome numbers could have been derived from pre-existing higher numbers. The cytological observations on all of the other mitotically unstable plants have been closely comparable to those on the 6*x* seedlings of Eldorado. Accordingly, it is considered that the same sequence of mitotic events occurs in all of these plants. Admittedly any sequence of mitotic events is hypothetical when it is based on observations of fixed material, since there is no way of knowing that each stage or phase gives rise to the next. However, the sequence presented gives a logical order for the configurations seen and does allow for the observed diminution of chromosome numbers.

The critical observations on which this sequence is based are:

1. At metaphase there may be two (or more) distinct plates of chromosomes each with its individual spindle (Figure 8 *F, G*). The total number of chromosomes in the two plates is equal to the number of chromosomes in some of the neighboring cells. Although it has been possible to make these chromosome number determinations with such precision in relatively few cases, it has always been strikingly apparent on the basis of the bulk of the chromosomes (cf. prophase and three groups of chromosomes in prometaphase in Figure 8*B*).
2. At anaphase there are four (or more) groups of chromatids with two spindles (Figures 8*H*, 9*A* and *B*).
3. At telophase there are four (or more) groups of chromatids with phragmoplasts evident at right angles to the orientation of the spindles and equidistant between the chromatid groups. Phragmoplasts have only been seen in the region where there was a spindle.

The stages of the cytological mechanism resulting in the decrease of chromosome numbers are considered to occur in the following sequence:

1. At prophase there is either an apparently single prophase nucleus or there is evidence of an irregular grouping of the chromosomes. In the example shown in Figure 8*A* there was evidence of two distinct groups. This stage is not considered critical because of the possibility that such apparent groupings are caused by the squash technique. However, the groups of chromosomes with independent spindles at metaphase and anaphase give no evidence of being caused by squashing.
2. The chromosomes at prometaphase are often more clearly seen in definite groups. For example, in Figure 8*B* three such groups are shown and in Figure 8*C* the more common occurrence of two distinct groups is illustrated. These groups were either of equal numbers of chromosomes or were quite unequal.
3. At metaphase there are usually two distinct plates of chromosomes as shown in Figure 8*D*. These plates are oriented in various ways. Some of the orientations are shown in Figures 8*D* (polar), 8*E* (one polar, one side view), 8*F* (two plates in side view and inclined), and 8*G* (two plates in side view and perpendicular). In order to conform to the terminology of Wilson *et al.*[16] and Vaarama[13] this stage is termed a split metaphase. It is used in the sense that the chromosomes of a single cell are separated into two distinct or split groups, each with its own spindle. There seems to be no

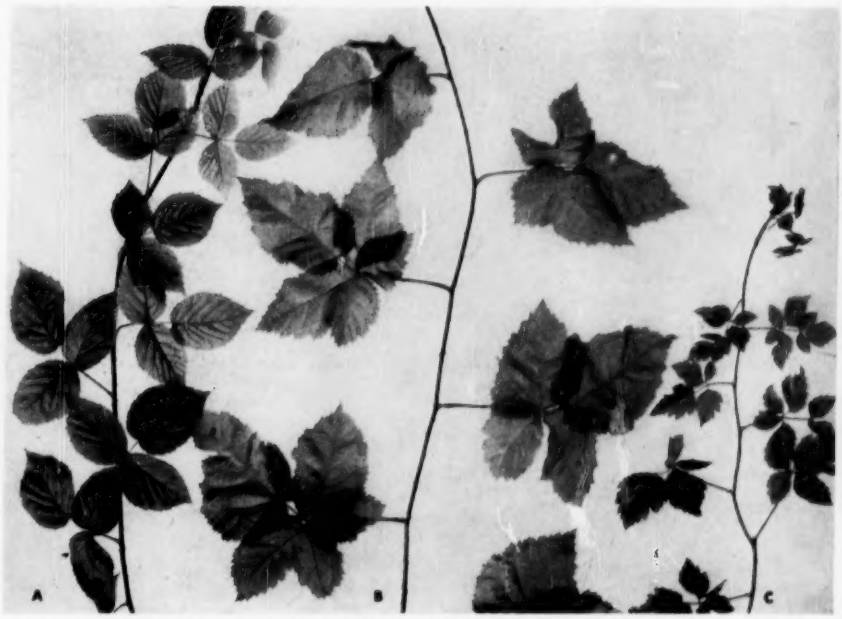
TABLE I. Parentage and numbers of mitotically unstable seedlings

Cross code number	Parentage	Numbers of mitotically unstable plants
56–49	Eldorado (4*x*) × self	5
56–53	Austin Thornless (8*x*) × (8*x*) Eldorado	1
56–56	Johnson (ca. 8*x*) × (8*x*) Eldorado	2
56–58	Wild seedling (ca. 6*x*) × (8*x*) Eldorado	1
56–61	Young (7*x*) × (8*x*) Eldorado	5
56–62	(8*x*) (Merton Thornless × Brainerd) × self	1
56–63	(8*x*) (Merton Thornless × Brainerd) × (8*x*) Eldorado	1
56–76	Cascade (9*x*) × (8*x*) Eldorado	2
56–79	Boysen (7*x*) × (8*x*) Eldorado	5
56–88	Hailsham (4*x*) Raspberry × Austin Thornless (8*x*)	5
	Total	28

TABLE II. Chromosome numbers from 32 root-tips of (6*x*) seedlings of Eldorado selfed*

Chromosome number	Number of cells
42	16
41	4
37	1
35	3
34	3
32	4
30	2
29	3
28	12
27	2
26	1
25	1
24	3

*Only those chromosome number determinations within an error of one.



THREE PRIMOCANES FROM A SINGLE MITOTICALLY UNSTABLE BLACKBERRY

Figure 7

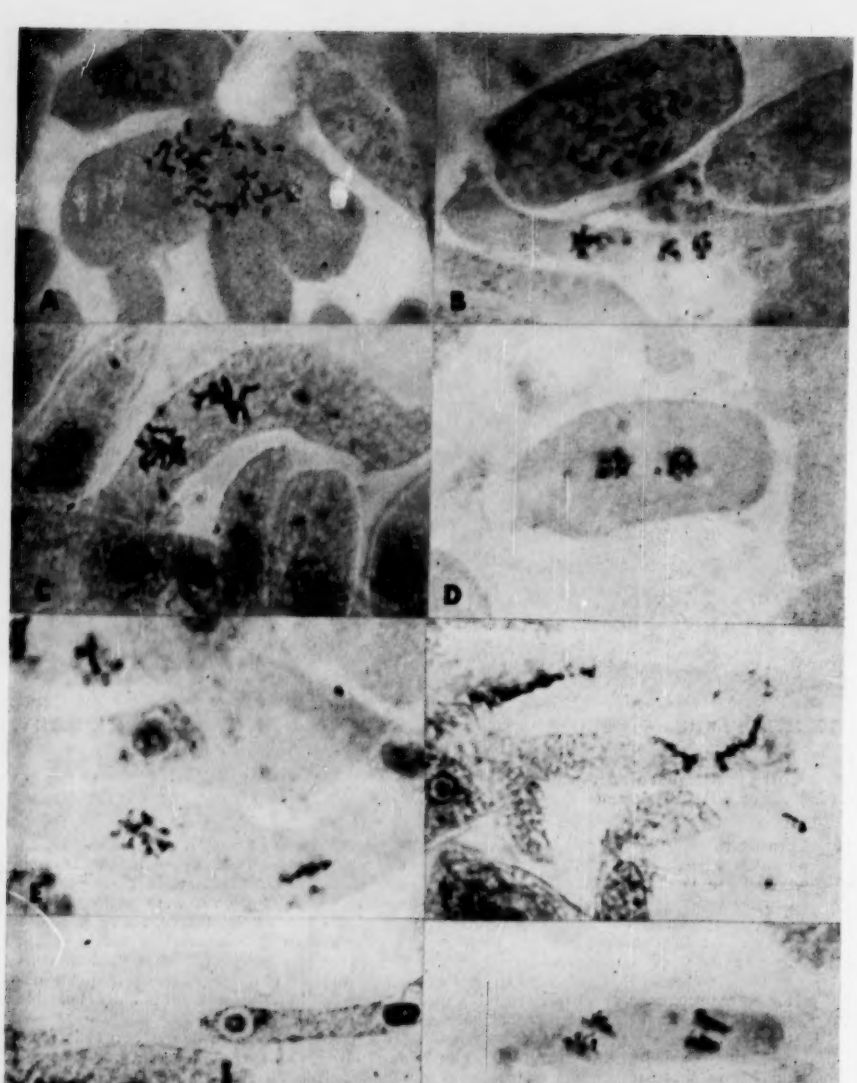
Three primocanes which originated as root suckers of a single unstable hybrid (Young × (8*x*) Eldorado). Primocanes are lettered from left to right, *A*, *B* and *C*. See text for details.

reason to consider the spindle to be split into two parts. The term "double-plate metaphase"[12] might have been used equally well.

4. A split anaphase follows the split metaphase. At this stage there are usually four groups of chromatids with two spindles (Figures 8*H*, 9*A* and *B*). In the more uncommon event of having three groups of chromosomes at prometaphase, there are then six groups of chromatids and three spindles at anaphase. The number of chromatids going to each anaphase pole is dependent on the number of chromosomes that were in each group of the split metaphase. The orientation of the spindles is dependent on the orientation of the plates at split metaphase.
5. At telophase phragmoplasts are evident between the groups of chromatids in the region of the spindles and *no* phragmoplast is formed between the original or split groups of chromosomes where there is no spindle (Figure 9*C*). After this stage there are formed three cells, two of which have a smaller chromosome number than at prophase and one cell which is binucleate. It should be noted that a telophase from a split metaphase such as shown in Figure 8*F* could have two groups of chromosomes in close proximity. If these groups were enclosed by a single nuclear membrane, then a partially binucleate or lobel nucleus would result. Binucleate cells (Figure 9*D*) and cells with lobed nuclei were of frequent occurrence in all the mitotically unstable plants.

Figure 9*G* and *H* illustrates two cells from one root-tip with very different chromosome numbers. In one cell there are 11 chromosomes and in the other 43. The root-tip was taken from a plant of the cross Cascade (9*x*) ($2n = 63$) × (8*x*) Eldorado ($2n = 56$). Chromosome number determinations made from axillary leaf-bud squashes, as well as root-tip squashes early in the life of the seedling showed most cells to have 59 ± 2 chromosomes. Each of the mitotic stages of split divisions was seen in this plant. Accordingly, the sequence of events leading to the decrease in chromosome numbers from 59 to 11 is considered to have occurred by the cytological mechanism outlined above.

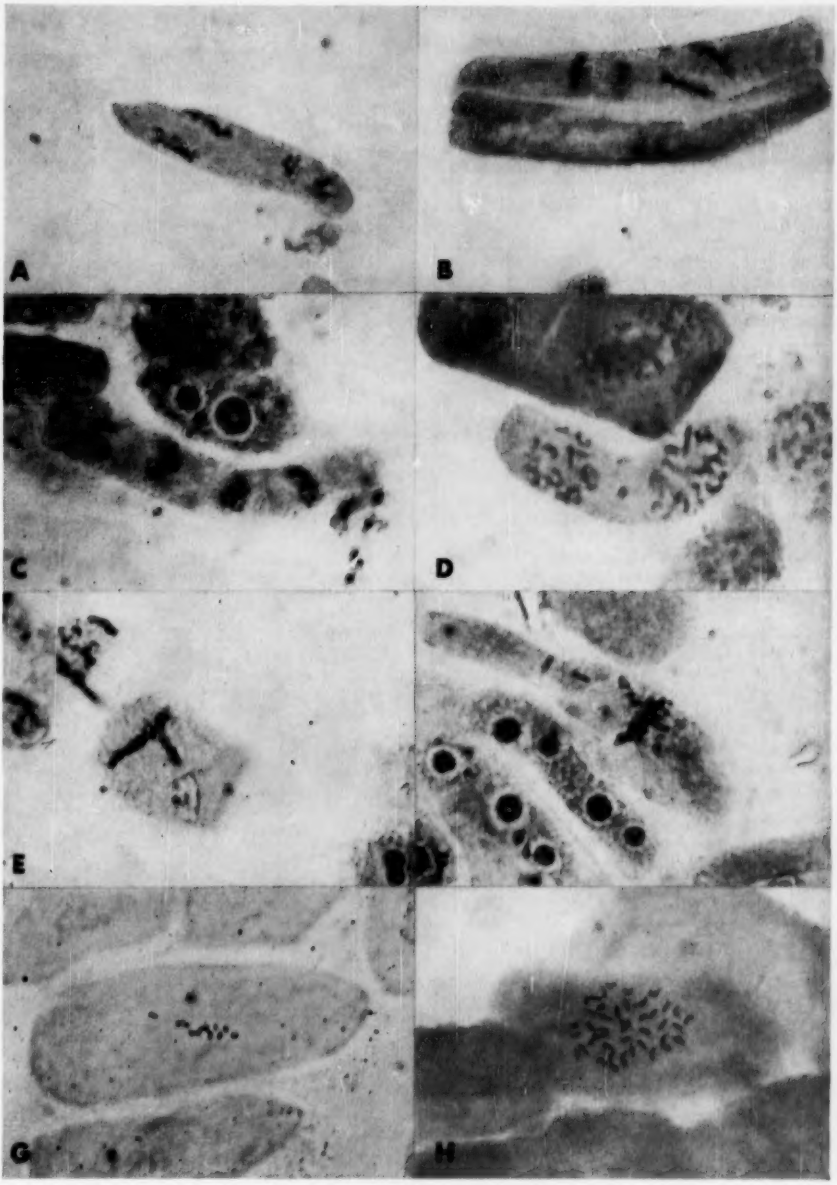
Most of the unusual configurations seen in mitotically unstable plants could satisfactorily



MITOTIC INSTABILITY

Figure 8

A—Late prophase with a suggestion of two groups of chromosomes. *B*—Prometaphase with three distinct groups of chromosomes and prophase with a high chromosome number. *C*—Prometaphase with two groups of chromosomes. *D*—Polar split metaphase. *E*—Split metaphase with one group in polar view and the other in side view. *F*—Split metaphase with two plates and two spindles in side view and inclined towards one another. *G*—Split metaphase with two plates and two spindles in side view and perpendicular to one another. *H*—Split anaphase. (Magnification 1000 ×.)



IRREGULAR MITOTIC DIVISION

Figure 9

A and *B*—Split anaphase. *C*—Split telophase. Note phragmoplasts across spindles and binucleate central cell. *D*—Prophase from a binucleate cell. *E*—Metaphase-"T"-shaped. *F*—Metaphase-"X"-shaped. *G*—Eleven chromosomes in cell from hybrid (Cascade × (8*x*) Eldorado). *H*—Forty-three chromosomes in a metaphase plate from the same root-tip as *G*. (Magnification 1000 ×.)

be classified in the afore-mentioned sequence of mitotic events. However, a few metaphases were seen which were "T"-shaped (Figure 9*E*), or "X"-shaped (Figure 9*F*) or even "V"-shaped. With configurations such as these, only one spindle (Figure 9*E*) in each cell has been seen. These observations differ from those of Vaarama[13] who classified these metaphases with split metaphases. The inability to effectively demonstrate the presence of two spindles in each cell in "T", "X", and "V"-shaped metaphases suggests that the separation of chromosomes into split groups is not complete in these configurations. These abnormal metaphases would be followed by anaphases with larger numbers of chromosomes at one pole than the other. A few such anaphases have been observed. This phenomenon would be an additional explanation for the variability in the chromosome numbers observed.

Most other features of the diminution of chromosome numbers presented in this paper are very similar to those described by Vaarama[13]. In both studies comparable mitotic stages including separate or split spindles were found, whereas no multipolar spindles were seen. However, Vaarama[13] concluded that it was the colchicine which was responsible for the upset in the spindle mechanism even in the second generation (C_2). That colchicine is not necessary to cause mitotic instability is evident, since the (6*x*) Eldorado seedlings reported here were from a progeny of untreated selfed Eldorado. A further difference between these studies is that many binucleate cells were seen in the present work. The presence of many binucleate cells obviates for *Rubus* Vaarama's postulate for *Ribes* that "Evidently cell wall formation is in principle similar to tetrad formation after the second meiotic division." Cell walls were formed only at right angles to the spindles, and were not formed between split plates where there was no spindle.

Another point of variance between these studies concerns the interpretation of the presence of lobed or constricted nuclei. Vaarama suggests that these are evidence for a certain degree of autonomy of nuclear parts and that they arise from single resting nuclei. However, two split metaphase plates which are inclined towards each other (Figure 8*F*) could give rise to a lobed nucleus at telophase (Huskins and Cheng, Figure 6*I*[7]). Accordingly, lobed nuclei in this study are considered to result from split divisions rather than to represent forerunners of them.

Snoad[11] found cells with two distinctly separate spindles in *Hymenocallis*, as well as tripolar spindles. He terms these phenomena "spindle abnormalities." This would seem to be an error in emphasis when considering two separately organized spindles which are perfectly normal in appearance and function, as seems to be the case in *Rubus*.

Menzel and Brown[9] discuss mosaic formation and somatic reduction in *Gossypium* and conclude that one possible mechanism for a common cause of these phenomena would be "some type of atypical segregational mitosis." Mosaic plants were found by them between the 4*x* ($2n = 52$) and 8*x* ($2n = 104$) levels of ploidy, but not at the 2*x*—3*x* levels (Table 2, *loc. cit.*) In *Rubus*, plants exhibiting mosaicism were found also, and all these plants were mitotically unstable. This instability was found to occur above the $2n = 42$ (6*x*) level. Menzel[8] reports another case of somatic reduction in a mosaic-forming hybrid of *Gossypium*. Here the somatic chromosome number had decreased from 78 to 69. It is probable that the same general phenomenon is involved in both *Gossypium* and *Rubus*.

Wilson *et al.*[16] have described variations of mitosis in both treated and untreated onion root-tips. The unusual divisions found by them in untreated root-tips closely approximate those reported here in *Rubus*. The mechanism causing the spontaneous reduction of chromosome number reported by Huskins and Cheng[7] could be interpreted as being of a similar nature also.

It should be noted that Huskins[6], Huskins and Cheng[7], Wilson[14], Wilson and Cheng[15], and Wilson *et al.*[16] were all working with diploid plants in which a very low percentage of cells with decreased chromosome numbers were seen. These cells with sub-diploid numbers would be unlikely to divide again. In both polyploid *Gossypium* and *Rubus* many of the cells resulting from irregular mitoses are capable of further division. For example, in *Gossypium*, Menzel[8] found an entire mutant branch, and in the present study of *Rubus* over 100 plants have been propagated asexually that had chromosome numbers less than those of their respective mother plants. In *Rubus*, extremely low numbers of 10 and 11 chromosomes have been seen rarely, but as was pointed out, it has not been possible to obtain asexually propagated plants with such chromosome numbers.

The mechanism causing mitotic instability presented here is considered too inexact to use the terms "reductional" or "segregational". It is inexact in that all gradations, from two completely separate metaphase plates with separate spindles, to "T"-shaped metaphases with one spindle, to a single metaphase plate with one or two chromosomes off the plate, have been seen. However, when there are two separate spindles in a single cell, the end result is the production of cells with a lower chromosome number. It is only in this sense that the division is reductional. The fact that two groups of chromosomes separate from a single resting nucleus permits the possibility of the expression of some factors that were previously masked by dominance (i.e., segregation). However, when considering high (7*x*-8*x*) polyploids, the most likely result

would be the loss of factors still represented in the chromosomes retained. Thus, the majority of the genetic changes from the split divisions would be quantitative rather than a qualitative or segregational nature.

The lack of phenotypic differences in the plants propagated by leaf-bud cuttings in contrast to the great phenotypic differences between the plants propagated by root-cuttings may be explained by contrasting the complex structure of a vegetative shoot apex with the simple structure of a root apex. The vegetative shoot apex in *Rubus* consists of three histogenic layers. Each of these three layers is maintained by the division of 3-5 primary cells at the extreme apex[3]. Unless one or more of these primary cells undergoes a split division, the growing point will continue to produce cells with the same chromosome number. The axillary leaf-bud primordia are in close proximity to the apical dome and originate from very few cells. Consequently there are few cells in this restricted area in which it is possible for split divisions to occur, in contrast to the multitude of dividing cells below the apex of the dome. Split divisions occurring later in the ontogeny of leaves have caused the formation of mosaic patterns and in several cases, of a whole leaflet of different morphology. On the other hand, the roots are a many branched system having only one histogenic layer. The secondary roots in this system arise adventitiously from the primary root. Split divisions occurring in the primary root will give rise to isolated groups of cells with lower chromosome numbers than the zygotic number. If then, secondary roots arise from isolated groups, the lower chromosome numbers would be perpetuated. Further split divisions and branching of roots would result in further decreases of chromosome numbers.

Thus from mitotically unstable plants it has been possible to isolate and propagate aberrant forms by means of root-cuttings that would not be apparent from propagation by leaf-bud cuttings. It follows that leaf-bud propagation is a more reliable means of propagating unstable clonal material than are root-cuttings.

Mitotic instability is a phenomenon that causes difficulties for the plant breeder who may wish to maintain a naturally occurring polyploid such as a (6*x*) seedling of Eldorado, or an artificially induced polyploid such as (8*x*) Eldorado. Since the canes are biennial in *Rubus*, it is necessary to propagate such unstable plants as (8*x*) Eldorado by leaf-bud cuttings each year in order to have any hope of maintaining the desired level of ploidy for crossing in the following year. The difficulty of maintaining certain high polyploids at a known level of ploidy has proved to be a real one in the program at Maryland. It has been necessary to continually propagate and reexamine cytologically many of the polyploids that are used in the breeding program.

It is extremely difficult to state with any assurance that one *Rubus* plant is completely mitotically stable and another is not. If a large number of split divisions are found it is assumed that the plant is mitotically unstable. However, if no split divisions are found and the chromosome number appears to be constant, there is always the possibility that insufficient observations have been made. Nevertheless, certain of the progeny from the crosses presented in Table I appeared to be quite stable, although considerably above the 6*x* level of ploidy. The genetic constitution necessary for mitotic instability, or conversely for mitotic stability, is not evident. However, one factor involved may be the degree of autopolyploidy of the unstable plant. This is indicated by the facts that (4*x*) Eldorado is mitotically stable, (6*x*) seedlings of selfed Eldorado are unstable, and (8*x*) Eldorado is markedly unstable; whereas apparently an allopolyploid variety[4] — such as Cascade (9*x*) is stable. In this connection one might speculate whether this is the reason that no eastern erect blackberries occur naturally above 4*x* in ploidy, whereas the western trailing blackberries are found to the 12*x* level of ploidy.

Many of the crosses in Table I had one parent which was an artificial polyploid, such as (8*x*) Eldorado. It should be noted that the nature of the breeding program was such that (8*x*) Eldorado and other artificial polyploids were involved in many crosses. Accordingly, it is not possible to say that artificial polyploids are the ones most likely to be involved in the production of unstable plants. Nevertheless, it seems that the plant breeder should be particularly watchful for mitotic instability when working with synthesized polyploids.[8, 9, 10]

Summary

Twenty-eight mitotically unstable seedlings were found among 10 progenies of *Rubus*. The number of chromosomes in some cells was found to range down to a number less than the gametic number of either parent. The essential feature of the mechanism of this diminution was the grouping of the chromosomes of one cell into two plates, each with its own spindle at metaphase. Studies by others indicate that a decrease of chromosome number is not peculiar to this genus. Similar, or perhaps identical phenomena have been described for *Ribes*, *Hymenocallis*, the *Triticinae* and *Gossypium*. However, mitotic instability in *Rubus* is more than a cytological anomaly, as is shown by the results of asexual propagation. Root-cuttings have given rise to plants with differing phenotypes. Cytological studies of these plants showed that they had low chromosome numbers which were usually aneuploid and that they were mitotically stable. On the other hand, leaf-bud cuttings produced plants with a similar phenotype to the mother plant and perpetu-

ated the mitotic instability. The differences between the results from root-cutting propagation and leaf-bud cutting propagation are explained by the contrasting histogenesis of roots and shoots.

Although colchiploid parents are not necessary to cause mitotic instability in the seedlings, it appears that they are often involved. The decrease in chomosome number of mitotically unstable *Rubus* stocks is a plant breeding problem which may be overcome by annual asexual propagation of the top of the plant by leaf-bud cuttings.

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◆

WHO CAN HELP?

ONE of the glaring errors committed in the writing of my book PRINCIPLES OF HUMAN GENETICS is the statement that Charles Darwin and Abraham Lincoln were the products of cousin marriages. Neither of them were, though Darwin, himself, married a cousin and Lincoln and Darwin both were born on February 12, 1809. I should like to substitute for the names of these two great men those of men of comparable distinction who are truly products of cousin marriages. No striking examples are known to me. Who can help?

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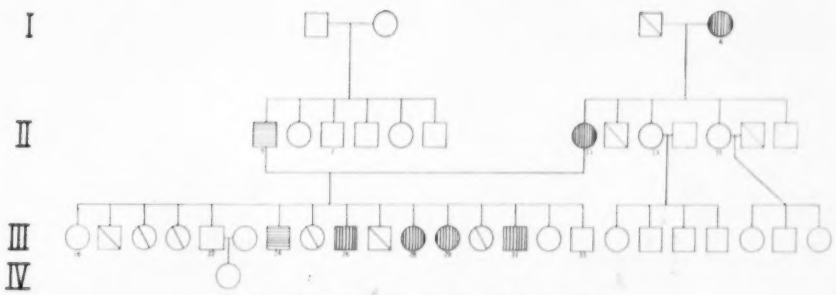
BRACHYTELEPHALANGY AND BRACHY-MESOPHALANGY IN THE SAME FAMILY

EMMANUEL MARGOLIS, ARMIN SCHWARTZ AND RAPHAEL FALK*



TWO DEFECTS IN THE SAME FAMILY
Figure 10

The horizontal lines within squares indicate brachytelephalangy, transmitted through the father. The vertical lines within squares and circles indicate brachymesophalangy, transmitted through the mother. The diagonal lines within squares and circles were either dead or not tested.

IN a sibship of 15 members (Figure 10) two separate hereditary traits could be definitely distinguished; brachytelephalangy transmitted through the father and brachymesophalangy transmitted through the mother. Such a mating appears to be very rare and this perhaps explains why not a single case was found reported in the literature available to us. We therefore think it is of sufficient interest to report it here.

The Line of Brachytelephalangy

The trait is characterized by the appearance of a broad, short, thick first, third and fourth finger, bearing a short, broad nail (Figure 11*A*).

The X-ray confirms (Figure 11C) that the seat of the anomaly of the affected finger lies in the terminal phalanx which appears broad, short, plump and shows a prominent flare at the tip.

Among the members of the present family, seen by the authors, this trait appears for the first time in both hands of the father—II-5 of the pedigree. Members II-6, 8, 9, 10 being in another country could not be examined. They are reported to be normal but we cannot rely on this information. II-7 was examined and was found normal.

The anomaly appears only once again in this family in a son, III-24, of the affected father. The appearance here is very similar indeed, but while in the left hand of the son the same three fingers (first, third and fourth) are affected, in his right hand only the third and fourth finger are shortened (Figure 11*B* and *D*).

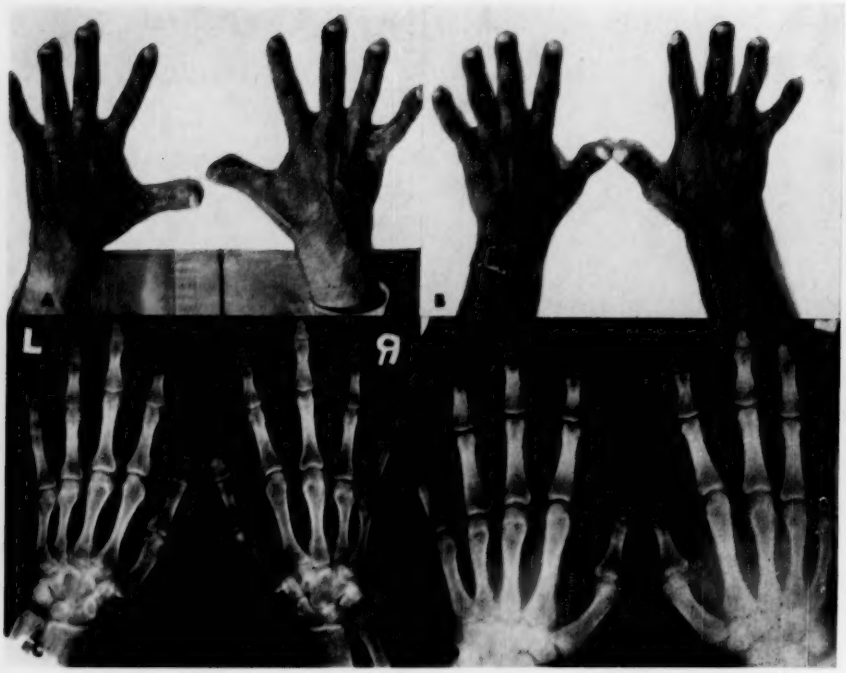
In the son another anomaly could be detected, namely an accessory bone near the pisiforme with (compensatory?) shortening of the processus styloid ulnae. This member of the family also suffers from epilepsy.

The inheritance from father to son indicates the dominant character of the trait. There is no significant deviation among the living siblings from the 1 : 1 ratio: 6 unaffected: 1 affected.

$x^2 = 2.28$; df = 1; P = 0.2-0.1.

Only if we suppose that all dead chil-

* Hadassah University Hospital, Jerusalem, Medical Outpatients Dept. "C"; X-Ray Department; and the Hebrew University, Department of Zoology, respectively. Thanks are due to Drs. Goldschmidt and E. Davis for their valuable criticism.



BRACHYTELEPHALANGY

Figure 11

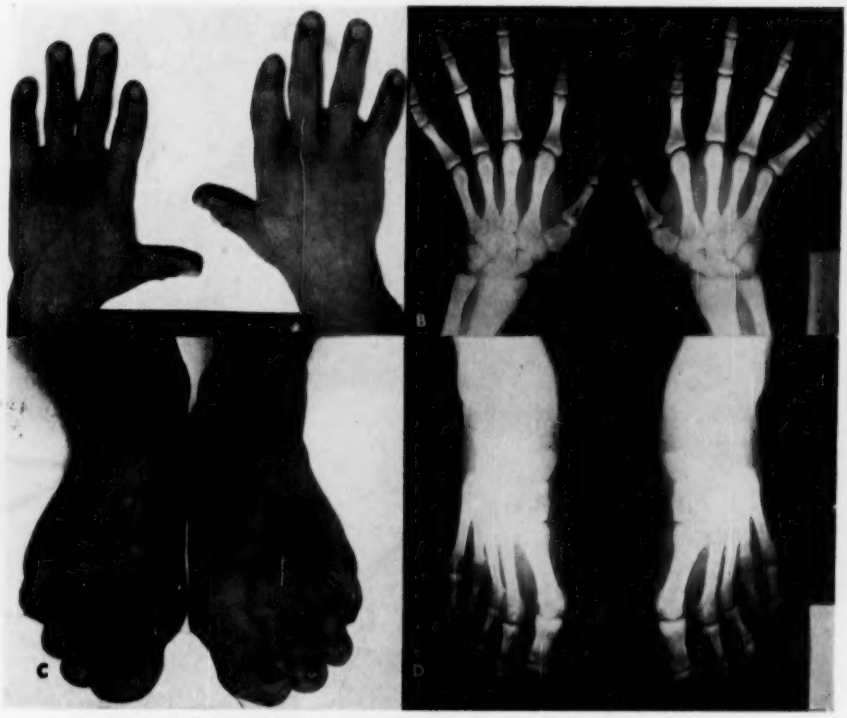
A and *C*—The first, third and fourth fingers of both hands of individual II-5 are shorter than normal, with a broad nail. The X-ray (*C*) shows the broad, short and plump terminal phalanx with a prominent flare at the top. *B* and *D*—In the left hand of III-24 of the pedigree the first, third and fourth fingers are affected with brachytelephalangy. In the right hand, only the third and fourth fingers are affected.

dren were unaffected is the deviation significant.

The Line of Brachymesophalangy

This trait is characterized by the appearance of short, otherwise normal fingers. The X-ray photographs in Figure 12 demonstrate that it is the middle phalangeal bone which causes the shortening. The phalanx is cubically shaped, markedly shorter than the normal.

In the present pedigree the trait was seen for the first time in the grandmother, I-4, and was transmitted to her daughter, II-11, without any variation in expression. In both cases the second and the fifth finger are affected, and the base of the proximal phalanx of the second finger is oblique (Figure 13*A* and *B*). However, in the other affected members marked variation in expressivity of the trait can be noted and it also appears with other bone abnormalities. In III-26 of the pedigree these additional abnormalities are abundant: the first carpal bone in both hands appears shorter than normal (brachymetacarpy, Figure 13*A* and *B*); the fourth toe in both legs is shorter than normal due to marked shortening of the fourth metatarsal and close fusion of the second and third phalanges of the fourth toe, which therefore appears with only two phalanges (Figure 13*C* and *D*). There is a cervical rib on both sides and generally stunted growth. All epiphyseal

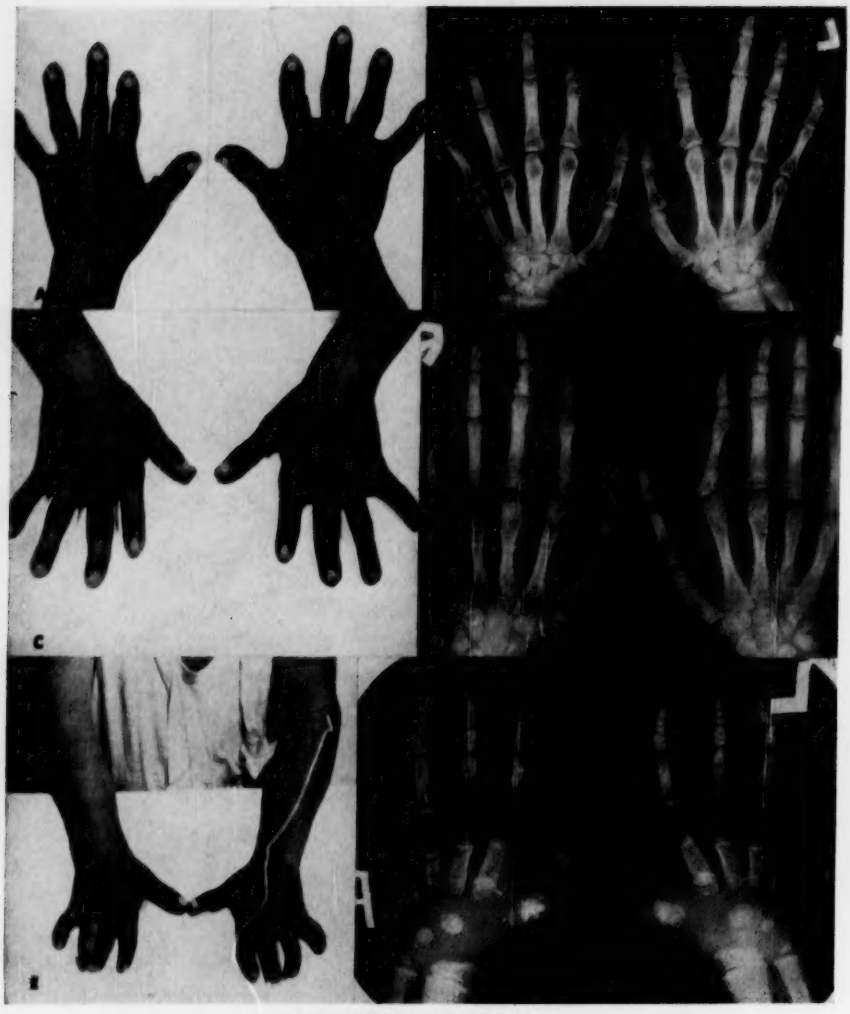


BRACHYMESOPHALANGY AND BRACHYMETACARPY IN III-26

Figure 12

A and *B*—In addition to the brachymesophalangy affecting the second and fifth finger in both hands, a short first metacarpal is to be noticed bilaterally. *C* and *D*—Shortening of the fourth metatarsal and fusion of the second and third phalanges of the fourth toe can be observed in the same individual.

lines of the fingers are closed, that of radius and ulna are open but narrowed. It is difficult therefore to draw a definite conclusion about bone age. In III-28 of the pedigree the brachymesophalangy affects not only the second and fifth fingers but also the third finger. In addition the epiphyses of the first phalanges of the right and left second fingers are larger than usual with an oblique base. The short middle phalanx of the second and fifth finger of the right hand and of the second, third and fifth finger of the left hand are without epiphysis whereas the middle phalanx of the right third finger has an irregular epiphysis with a narrowed epiphyseal line and is only slightly sorter than normal (Figure 13*C* and *D*). In III-29 only a slight degree of brachymesophalangy of the second and fifth finger bilaterally is to be seen. The epiphyseal line of the short middle phalanges of the second finger is centrally obliterated in both hands. The first metacarpal is somewhat short. In III-31 brachymeophalangy of the second, third, fourth and fifth finger in both hands is to be noted, the epiphyses of the short middle phalanges being absent. There are accessory ossification centers present at the proximal ends of the second and fifth metacarpals in both hands and the epiphysis of the first metacarpal in the left hand is absent. The first metacarpal in both hands is short and broad with



BRACHYMESOPHALANGY

Figure 13

A—The second and fifth fingers in both hands of individual II-11 are affected with brachymesophalangy as shown in the X-ray, *B*. The short index is to be easily distinguished; the fifth finger, however, appears almost normal in length. *C* and *D*—Not only the second and fifth but also the third finger of individual III-28 is affected. The epiphysis of the first phalanx of the second finger bilaterally is larger than usual with an oblique base. The short middle phalanx of the second and fifth finger of the right hand and the second, third and fifth finger of the left hand are without epiphysis whereas the middle phalanx of the right third finger has an irregular epiphysis with a narrowed epiphyseal line. *E* and *F*—The second, third, fourth and fifth finger in both hands of individual III-31 are affected. The epiphysis of the short middle phalanges as well as the epiphysis of the first metacarpal in the left hand are absent. The first metacarpal in both hands is short and broad with coarse irregular structure. Accessory ossification centers are present at the proximal ends of the second and fifth metacarpals in both hands. In the carpus only two ossification centers could be seen (compatible with bone age of two years, the real age of the boy being seven years).

coarse irregular structure. In the carpus only two ossification centers (triquetrum and capitate) could be seen, which is compatible with bone age of two years (Figure 13*E* and *F*); the well developed epiphysis of ulna and radius, however, point to bone age of seven years which is also the chronological age of the child. This member of the family shows also clinical and roentgenologic evidence of congenital heart disease.

From the above description it is obvious that we are dealing with a trait which shows variations in expressivity since the sibship of the third generation may be affected either more or less severely than their mother, II-11, and their grandmother, I-4. The trait apparently has good penetrance.

Among the living tested relatives of I-4 the ratios are as follows:

Generation II	1 aff.	: 2 unaff.
Generation III	4 aff.	: 3 unaff.
	5 aff.	5 unaff.

The ratio would be significantly disturbed only if there were unproportionally many affected or unaffected among the dead and non-tested individuals.

Discussion

It is of interest that the mating of two individuals with the deformities described above did not result in a living child being affected both with brachy-telephalangy and -mesophalangy. Every child has ½ a chance to inherit the mother's defect and ½ a chance to have the father's defect, i.e. ¼ of a chance to get both defects (if full penetrance of both defects is assumed). The likelihood that no living child will be doubly affected is $(1-¼)^7 = 0.134$, i.e. such an event can be due to chance in only 13-14 such families out of a hundred.

There are two possible hypotheses to explain the genetic background of these two bone defects: according to the first there is a series of multiple alleles in one locus, one responsible for brachymesophalangy, one for brachytelephalangy and the third for normal phalanx formation. According to the second hypothesis brachytelephalangy and bracymesophalangy are caused by two genes situated in different loci on the chromosomes. The reported pedigree does not permit a conclusive decision concerning the two hypotheses. Each hypothesis allows three alternative explanations for the absence of the doubly affected phenotype, as shown below:

A. Mulitple Allelism

1. Lethality of the compound heterozygote.
2. Dominance of one "defective" gene on the other. (Brachymesophalangy)
3. Partial penetrance and/or expressivity of one and/or the other defect.

B. Genes at Different Loci

1. Lethality of the double heterozygote—"synthetic lethal."
2. Epistasis of one "defective" gene on the other (Brachymesophalangy)
3. Partial penetrance and/or expressivity of one and/or the other defects.

1. If the non-living children and/or the children to be born from the pregnancies which terminated in abortion had bone and skeletal malformations of some degree of severity, it seems possible that they may have been individuals having both brachytelephalangy and brachymesophalangy, the defects in them being so severe that they are lethal. Mohr and Wriedt[2] described a family with brachymesophalangy where inter-marriage of two affected persons resulted in a very severely malformed child who died after one year. They believe that this child was the homozygote for the brachymesophalangy gene. No similar case is known concerning brachytelephalangy, but it was repeatedly noted that the lack of reports on homozygotes for many other "dominant genes" (Levit[1]) in human beings may indicate the lethality of these genes in double dose. It appears therefore possible that the double-affected zygotes in the family studied are lethal. However, in the absence of reliable and accurate data concerning the dead children this assumption cannot be proved.

2. Alternatively as there is only one child affected by brachytelephalangy compared to four children affected by brachymesophalangy in the third generation one has to consider the possibility that brachymesophalangy is dominant over (according to hypothesis *A*) or epistatic (according to hypothesis *B*) over brachytelephalangy.

3. In the literature variations in penetrance and expressivity of such defects are described. Such a variation is found also in the expressivity of brachytelephalangy in this pedigree, and it is possible that the deviation from the expected number of defective individuals is due to incomplete penetrance.

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THE GENETICS OF SELF-INCOMPATIBILITY IN THE RADISH

D. R. SAMPSON*

STOUT[1] found the radish (*Raphanus sativus*) to be self-incompatible. Sears[3] discovered that the immediate cause of incompatibility in the radish was the failure of incompatible pollen tubes to penetrate the stigmatic surface.

The genetics of self-incompatibility in radishes was investigated by A. G. Brown at the John Innes Horticultural Institution in 1944 and 1945. He was unable to interpret the data. Bateman[2] published one part of this work, the sib compatibility relationships in a family resulting from a single cross. Two intra-incompatible but inter-compatible groups were present. Bateman regarded this as the expression of a sporophytic incompatibility system. The present study confirms Bateman's interpretation.

Materials and Methods

During the winter of 1954-55 plants of the annual radish, "Scarlet Globe," were grown in the greenhouse. Crosses and selfings were made with fresh stigmas and pollen. The resulting number of mature fruits was used to indicate compatibility relationships. On the average, compatible pollinations gave 73 percent fruit set, and incompatible pollinations gave 5 percent set.

Three self-incompatible plants, designated, 1, 2 and 4, were chosen to be the parents of future populations. Parents 1 and 2 were mutually cross-incompatible in both directions. The crosses 1×4, 4×1 and 2×4 were compatible. Cross 4×2 gave two fruits from 10 flowers pollinated.

The results based on fruit set were checked by the microscopic examination of stigmas squashed in aceto-carmine 24 hours after pollination[3]. For this method, four flowers were pollinated and the four stigmas were squashed on one slide. If 10 or fewer unstained grains with pollen tubes (compatible grains) were found on the slide, the pollination was scored (—); if from 11 to 50, the results were noted (±); if over 50, (+). Plants 1, 2 and 4 were self-compatible, by the stigma squash method; 1 and 2 were mutually cross-incompatible, but 4 was cross-compatible with 1 and 2 in both directions. This and later findings indicate that the low fruit set from the cross 4×2 was due to the weak vegetative condition of parent 4.

Seven progeny families were studied during the summer of 1955. These plants were grown in an insect-free screen house. Compatibility determinations were made entirely by the stigma squash method. The results are recorded on the accompanying checkerboard diagrams (see Figures 14 and 15). Females are entered at the top of a diagram, males at the side. The first column and row of a diagram contain the allele subscripts of the *S*-genotype proposed for an individual or class. An active allele is denoted by a dot superscript, a recessive allele by parenthesis, and incomplete recessiveness by dot and parenthesis. The second column and row contain the plant numbers; 143, for example, designates plant 3 from the cross 1×4. The prefixed family numbers are usually omitted from sib-compatibility diagrams. None of the parents 1, 2 or

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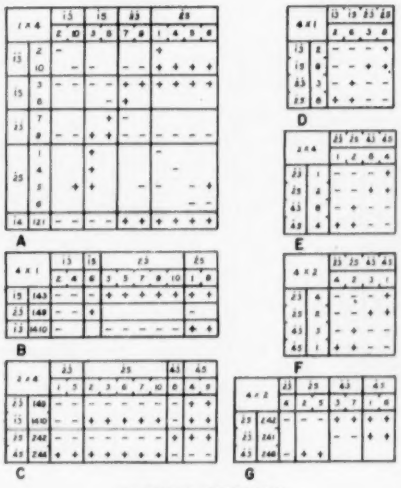

FAMILY GROUPS

Figure 14

A—Sib-compatibilities in family 1 × 4, and the reaction of the pollen of plant 121 toward this family. *B*—Division of family 4 × 1 into four classes by the pollen of appropriate testers. *C*—Division of family 2 × 4 into four classes. *D*—Sib-compatibilities in family 4 × 1. *E*—Sib-compatibilities in family 2 × 4. *F*—Sib-compatibilities in family 4 × 2. *G*—Division of family 4 × 2 into four classes. See text for details.

4 was tested against progeny, nor entered in the diagrams.

Results and Discussion

Family 1 × 4

Analysis of the progeny families began with 10 plants of family 1 × 4. First, all plants were selfed and all were found to be self-incompatible. Then plant 143 was chosen at random and its pollen was used on all sibs; this gave two groups. Later, the behavior of the pollen of plant 121 confirmed this division into two groups. Random sib pollinations were then used to detect further groups. The pollen of 149 and 1410 completed division of the family into four groups (Figure 14*A*). One pollination disagreed.

The sib-compatibility pattern of family 1 × 4 is characteristic of a sporophytic incompatibility system whereby the phenotype of the pollen is determined by the genotype of the pollen parent. Accordingly, the reactions of the four groups were arranged on a checkerboard to form a compatible diagonal[1] and the genotypes S_1S_3, S_1S_4, S_2S_3 and S_2S_4 were assigned to the appropriate groups (Figure 14*A*). Phenotypes could then be deduced and it was concluded that S_1, S_2 and S_3 were active in the pollen and stigmas wherever present, but that S_4 was recessive in pollen or stigma or both.

In addition, the behavior of the pollen of 121 toward the classes of family 1 × 4 showed that S_1 was active in this pollen. Since 121, a product of pseudo-compatibility, possessed one of the three active alleles of family 1 × 4 (Figure 14*A*), it followed that S_1 was present in parent 1. Accordingly parent 1 must have been S_1S_2 and parent 4 must have been S_3S_4.

Most of the remainder of this study was devoted to testing the foregoing interpretation of family 1 × 4. The following predictions were based on this interpretation: (a) the reciprocal 4 × 1 should contain the same four classes as 1 × 4; (b) families 2 × 4 and 4 × 2 should contain S_3 and S_4 as alternate alleles from parent 4; (c) parent 2, which was cross-incompatible with 1, should have S_1 or S_2 and thus families 2 × 4 and 4 × 2 should have plants with S_1 or S_2 and, consequently, (d) some of the genotypes found in family 1 × 4 should be recovered from 2 × 4 and 4 × 2. All predictions were realized.

Family 4 × 1

Ten plants of family 4 × 1 were divided into four groups by the pollen of 143 (S_1S_3), 149 (S_2S_3) and 1410 (S_1S_4) (Figure 14*B*). One member of each group was entered in sib combinations and the resulting compatibility pattern was the same as in family 1 × 4 (Figure 14*D*). These results were exactly as expected.

Family 2 × 4

Ten plants of family 2 × 4 were tested with the pollen of 149 (S_2S_3) and of 1410 (S_1S_4). Three groups appeared (Figure 14*C*). One group had S_3 active in the stigma, the largest group had S_4 active in the stigma and the third group had neither S_1, S_2 nor S_3. The reaction of the largest group showed parents 1 and 2 must have had S_3 in common. Further analysis of family 2 × 4 used the pollen of 242 and 244 and gave four groups. One member of each group was used in sib crosses (Figure 14*E*).

Genotypes were assigned to the groups of family 2 × 4 (Figure 14*E*) by using the compatible diagonal method of Bateman and the data in Figure 14*C*. S_1, S_3 and S_4 were active wherever present in this family, but S_2 was inactive in pollen or stigma or both.

Family 4 × 2

With a sporophytic incompatibility system, family 4 × 2 should segregate for the four groups found in 2 × 4. Pollen of 241 (S_2S_4), 242 (S_2S_3) and 248 (S_4S_4) split the seven plants of this family into the expected groups (Figure 14*G*). One plant from each group was entered in sib combinations (Figure 14*F*) and the resulting pattern was, as expected, the same as that obtained in family 2 × 4.

Inter-family		i3	i5	23		25		43	45	i2	i4	24	22
		1410	143	149	241	141	242	248	244	211	121	122	223
i3	1410	−	−	−	−	+	+	−	+	−	−	+	+
i5	143	−	−	+	+	+	+	+	+	−	−	+	+
23	149	−	+	−	−	−	−	−	+				
	241	−	+	−	−	−	−	−	+	−	+	−	−
25	141	+	+	−	−	−	−	+	+				
	242	+	+	−	−	−	−	+	+	−	+	−	−
43	248	−	+	−	−	+	+	−	−	+	−	−	+
45	244	+	+	+	+	+	+	−	−	+	−	−	+
i2	211	−	−	−		−		+	+	−	−	−	−
i4	121	−	−	+		+		−	−	−	−	−	+
24	122	+	+		−		−	−	−	−	−	−	−
22	223	+	+	−		−		+	+	−	+	−	−

A

242×143		i2			2(5)			i(5)			55			
		1	7	13	3	5	6	2	9	10	4	8	11	12
i3	1410	−	−	−	+	+	+	−	−	−	+	+	+	+
23	149	−	−	−	−	−	−	+	+	+	+	+	+	+
55	4	+	+	+	+	+	+	±	±	+	−	−	−	−

B

242×143 & 1×4		i2	2(5)	i(5)	55	i3	i(5)	23	2(5)
		1	5	2	4	1410	143	149	141
i2	1	−	−	−	+	−	−	−	−
2(5)	5	−	−	+	+	+	+	−	−
i(5)	2	−	+	−	+	−	−	+	+
55	4	+	+	±	−	+	+	+	±
i3	1410	−	+	−	+				
i(5)	143	−	+	−	+				
23	149	−	−	+	+				
2(5)	141	−	−	+	+				

C

2×1 & 1×2		i2	24	i4		
		211	122	121	123	124
i2	211	−	−	−		
24	122	−	−	−	−	−
i4	121	−	−	−		
45	244	+	−	−	−	−
23	241	−	−	+	+	+
i5	143	−	+	−	−	−

D

INTER-FAMILY COMBINATIONS

Figure 15

A—Inter-family combinations involving the 10 genotypes available in 1955. *B*—Division of family 242 × 143 into four classes. *C*—Pollinations involving representative genotypes from families 242 × 143 and 1 × 4. *D*—Analysis of families 2 × 1 and 1 × 2.

Family 2 × 2

Since parents 1 and 2 had S_2 in common, and since S_3 and S_4, segregated as alternative alleles in family 2 × 4, it followed that parent 2 was S_2S_4. Family 2 × 2 contained five plants, four obtained by bud pollination and one by pseudo-compatibility. These plants were analyzed by the pollen of 241 (S_4S_5) and 244 (S_4S_5); 223 and 225 (S_5S_5) were incompatible with 241 but were compatible with 244; 221, 222 and 224 (S_2S_4) were incompatible with both testers. This confirmed the proposed genotype of parent 2.

Families 2 × 1 and 1 × 2

Family 2 × 1 contained one plant; 1 × 2 contained four. All were the result of pseudo-compatibility. The genotypes S_1S_2, S_1S_4, and S_2S_4 were realized when the five plants were tested with the pollen of 244 (S_4S_5), 241 (S_2S_5) and 143 (S_1S_5), (Figure 15*D*). All alleles were active so the five plants were mutually incompatible.

Inter-family Combinations

Five *S*-alleles have been proposed to explain the results from seven families derived from three parents. Nine of the 10 possible heterozygotes and one homozygote were recovered in the progeny families. The results from crossing representatives of each genotype are given in Figure 15*A*. Genotypes S_2S_3 and S_2S_4 were entered twice so that those from family 1 × 4 could be compared with those from family 2 × 4.

The results were in complete agreement with previous conclusions. S_1, S_2, S_3 and S_4 act fully and independently of each other in pollen and stigmas of heterozygotes. On the other hand, no activity could be ascribed to S_5 in heterozygotes but heterozygotes possessing S_5 were not tested against S_5 known to be active.

Family 242 × 143

To determine the activity of S_4, family 242 (S_2S_4) × 143 (S_1S_4) was analyzed during the spring of 1956. Representatives of family 1 × 4 were overwintered in the greenhouse to provide testers. The pollen of 1410 (S_1S_4) and 149 (S_2S_4) divided the 13 plants of the family into four groups (Figure 15*B*). One member of each group was entered in sib combinations and crosses with representatives of 1 × 4 (Figure 15*C*). Since plants of S_2S_4 constitution were self-incompatible, S_4 was evidently active in the homozygote. No activity of S_4 was detected in the pollen of S_1S_4 or of S_2S_4. S_4 was slightly active in the stigmas of two S_1S_4 plants and one S_2S_4 plant. Thus S_4 appears to be recessive to S_1 and S_2 in the pollen but i ompletely recessive in the stigma.

Summary and Conclusions

Self- and cross-incompatibility in the "Scarlet Globe" radish (*Raphanus sativus*) was found to be controlled by multiple oppositional alleles at one locus. The reaction of the pollen is sporophytically determined. Five *S*-alleles were recovered from three original plants. Four *S*-alleles acted fully and independently of each other in heterozygotes. The fifth *S*-allele was recessive in the pollen and incompletely recessive in the stigmas when tested in heterozygotes with two of the active alleles.

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◆

An Early Report of a Fertile Female Mule

J. T. Baldwin, Jr.*

Crew and Smith[1] stated that "however rarely it may happen that a mare mule may become a parent, this undoubtedly does take place occasionally." There are no records of fertile male mules.

The purpose of this communication is to call attention to an early report of a fertile mare mule that appeared in a journal not generally available to geneticists: *Farmers' Register*, an authoritative periodical edited and published monthly in Virginia from June, 1833, to December, 1842, by Edmund Ruffin.

In a letter-to-the-editor dated 2d May, 1834, John Thompson Kilby wrote that a mare mule owned by him had produced a male offspring during the previous month on his farm in Nansemond County, Virginia (*Farmers' Register* 2:3, 1834):

> A circumstance has occurred on my plantation, which seems to be against the general principles of nature. On the 23rd of April 1834, a female mule of mine had a colt, never suspected by me until I saw its birth. I had worked her hard all last year, upon the farm, and on the railroad, through the winter hauling marl, and all the month of March hauling logs, from a distance of two miles, six loads a day, and thirty logs each load, making twenty-four miles each day. She was at work all April hauling out manure, until the 23rd. On that day I had gone, a little before night, from the labors of the day, owing to one of my family being sick; and about 5 o'clock in the evening, the boy that drove the mule, came running to the house, saying that Jenny (for that was her name) had a colt. I went out and in a few minutes thereafter, the *thing* (for I know not what to call it) was delivered, and is now doing well. I never suspected the mother's being with foal, because I thought it contrary to nature, though I had for four or six weeks observed that her belly was enlarged, and so much so, that the cart had to be altered, as it rubbed her. She showed no other sign—so I did not suspect it. She has little or no bag, though I believe she gives a plenty of suck, as her *child* is now getting fat. At first it was very poor. Now you will ask what is the father of it? I cannot say—but believe a colt of mine, now three years old. He ran out on Sundays, with the mules, and the black boy tells me that there was cause for such an effect. So it is, the mule has a colt, and it is exactly like the young stallion. If this is a matter of curiosity,

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you may give publicity to it, under my name. Hundreds can prove the fact—and several can testify that they were present at the birth.

/Signed/ John Thompson Kilby

P. S. The mother certainly is a *mule*, she was foaled mine, and is now ten years old.

The owner reported that the colt died on 20th October, 1834, seemingly from lockjaw (*ibid.*, *2*: 389, 1834).

Then, on 17th September, 1835, Mr. Kilby wrote to the Editor: "Permit me to record in your *Register* the birth of a second mule colt of mine, on the 13th of August, 1835. The same mule brought a fine female colt, jet black, save a star in its forehead, and one foot white. It partakes, as did the other, more of the horse than of the mule, and is a much finer colt. It can be seen in my pasture by any and every one" (*ibid.*, *3*: 440-441, 1835). This second colt, sired "by a horse," died on 26th August, 1836; an autopsy showed "that every thing that had been given it was then in its stomach, and had never passed on to the bowels" (*ibid.*, 4: 357, 1836).

Could this female "mule" in truth have been a jennet? That she was named "Jenny" is basis for skepticism, certain though Mr. Kilby was that the "mother . . . is a *mule*." Her excellent record for work suggests that she was not an ass. And the fact that each colt resembled the horse father is evidence that the mother was not a jennet. This is in accord with most reports on fertile female mules: the offspring strongly resemble the father whether he be jackass or horse. If indeed the mother happened to be a female horse with mulelike aspect, the progeny should, of course, be of horse phenotype if the father were a horse. But if the father were an ass, the offspring should then be a mule: I know of no such records, for when a reputed mule has been mated to a jack the colt has been asinoid.

"Jenny," like a number of fertile mules in the literature—notably "Old Beck" (Groth[2]), foaled more than once. This causes some hesitancy in accepting such reports as valid: production of a functional egg in an interspecific hybrid should be exceedingly rare. Available data indicate that the *2n*-number of chromosomes for the horse, the ass, and their hybrid is *66* and that meiosis is irregular in the mule (Makino[3]). It should be understood, however, that the chromosome sets of certain mules might be more similar than those of others. For such individuals chromosome pairing would be more nearly complete; fertility would be higher. Moreover, once a mule has proved her fertility, the owner would be inclined in many instances to see that she were in the future auspiciously bred. Chances are that many potentially fertile mules never have the opportunity to be serviced at the right instance.

My opinion is that "Jenny" should be added to the list of mare mules that are considered to have been fertile. At any rate, the report by Mr. Kilby is a felicitous one and worthy of being read for its own sake.

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MORMONIELLA MERRY-GO-ROUNDS

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IN the wasp Mormoniella it is possible to make eye colors alternate in successive generations of inbreeding. Only one type of fertile male is produced each generation and this is alyays different from the type occurring in the preceding and the following generations. There are three types of females, one being the same in every generation. The other two types alternate. If the wasps are etherized after brother-sister matings have occurred and the females with the alternating eye color are selected and set for breeding the next generation, it is easy to keep this Merry-Go-Round going on indefinitely.

Figure 16 shows eight examples of such alternations of generations. Each example is based upon three mutant alleles of a single series, *R*. These genes are each designated by a symbol suggesting the eye color of the mutant type with a number or with the initial letters of the finder. In this way the specific mutation giving rise to the gene is designated (Figure 16, column 1). The genes of the *R* series, each segregating as a unit from its allele, are pleiotropic consisting of different factors affecting different processes. The formulae for these mutant genes (column 2) are expressed in terms of these factors which indicate impairment or diminution of wild-type functions brought about by the mutations. Each mutant gene causes impairment of one, two or three functions. The impairments act as recessives to the normal wild-type condition and hence the compound females may be wild type if the factors for impairment affect different functions (complementary allelism) or mutant type if they affect the same function (simple dominance or blending).

There are two subseries, *O* and *S*, of alternative factors affecting processes of eye-color development. Number of alternatives in each subseries is unknown and indefinitely large with differences in colors resulting from different mutations sometimes pronounced but often intangible. Subseries *O*, placed first in the gene formulae, ranges from wild-type brown, +, to colorless oyster white, *oy*. Subseries *S*, placed second in the gene formulae, ranges from brown, +, to scarlet, *st*. Similar names of factors in the two different subseries do not imply similar functions. An *O* dahlia, *da.*+, for example, is complementary with an *S* dahlia, +.*da*, producing wild-type "doubly heterozygous" compound females.

One of the three genes in each Merry-Go-Round has a lethal, *l*, or a male-sterile, *ms*, effect. Two of the Merry-Go-Rounds, F and H, include genes with factors causing female sterility, *fsa*. In each Merry-Go-Round formulae of the two types of compound females and of the haploid males of the "odd" generations are given (Figure 16, columns 3, 4 and 5) followed by formulae for the "even" generations (columns 6, 7 and 8). Under each formula is given the eye color determined by the genotype. The females retained for breeding always have the gene with the lethal, *l*, or the male-sterile, *ms*, factor.

In Merry-Go-Round A the three genes are oyster-801, peach-333.5 and scarlet-DR. Scarlet females appear in each generation. Tinged (peach-oyster compound) females alternate with wild-type brown (complementary allelism). Scarlet males alternate with peach. The gene peach-333.5 is doubly recessive for color factors, mutant both in *O* and in *S*.

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R alleles		Generations 1, 3, 5, 7, etc.			Generations 2, 4, 6, 8, etc.		
designations	formulae	females discarded	females retained	males	females discarded	females retained	males
A oy-801 pe-333.5 st-GR	oy. +.l pe.st.+ +.st.+	+.st.+ / pe.st.+ scarlet	oy. +.l / pe.st.+ tinged	+.st.+ scarlet	pe.st.+ / +.st.+ scarlet	oy. +.l / +.st.+ brown	pe.st.+ peach
B oy-816 pe-333.5 st-GR	oy. +.ms pe.st.+ +.st.+	+.st.+ / pe.st.+ scarlet	oy. +.ms / pe.st.+ tinged	+.st.+ scarlet oy.+.ms oyster	pe.st.+ / +.st.+ scarlet	oy. +.ms / +.st.+ brown	pe.st.+ peach oy.+.ms oyster
C vm-809 or-336.2 mh-819	+.vm.l.+ or.st.+.+ mh. +.+.C	mh.+.+.C / or.st.+.+ mahogany (black)	+.vm.l.+ / or.st.+.+ scarlet (lavender)	mh.+.+.C brown (black) +.vm.l.+ vermilion (lavender)	or.st.+.+ / mh. +.+.C mahogany (black)	+.vm.l.+ / mh. +.+.C brown (black)	or.st.+.+ orange (oyster) +.vm.l.+ vermilion (lavender)
D da-817 pe-333.5 oy-GR	+.da.l pe.st.+ oy. +.+	oy.+.+ / pe.st.+ tinged	+.da.l / pe.st.+ dahlia	oy.+.+ oyster	pe.st.+ / oy. +.+ tinged	+.da.l / oy. +.+ brown	pe.st.+ peach
E oy-805 st-GR da-GF	oy.st.l +.st.+ da. +.+	da.+.+ / +.st.+ brown (black)	oy.st.l / +.st.+ scarlet (oyster)	da.+.+ dahlia (lavender)	+.st.+ / da. +.+ brown (black)	oy.st.l / da. +.+ dahlia (lavender)	+.st.+ scarlet (oyster)
F st-474 oy-423 mh-819	+.st.l. + .+ oy.st.+.fsa.+ mh. +.+. + .C	mh.+.+.+.C / oy.st.+.fsa.+ mahogany (black)	+.st.l.+.+ / oy.st.+.fsa.+ scarlet (oyster)	mh.+.+.+.C brown (black)	oy.st.+.fsa.+ / mh. +.+. + .C mahogany (black)	+.st.l.+.+ / mh. +.+.+.C brown (black)	oy.st.+.fsa.+ oyster (oyster)
G st-474 oy-GR oy-RH	+.st.l oy. +.+ oy.st.+	oy.st.+ / oy. +.+ oyster (oyster)	+.st.l / oy. +.+ brown (black)	oy.st.+ oyster (oyster)	oy. +.+ / oy.st.+ oyster (oyster)	+.st.l / oy.+.+ scarlet (oyster)	oy.+.+ oyster (oyster)
H st-474 st-669 oy-423	st.st.+.fsa +.st.+.fsa oy.st.+.fsa	st.st.+.fsa / +.st.+.fsa scarlet	+.st.l.+ / +.st.+.fsa scarlet	oy.st.+.fsa oyster	+.st.+.fsa / oy.st.+.fsa scarlet	+.st.l. + / oy.st.+.fsa scarlet	+.st.+.fsa scarlet

EIGHT MERRY-GO-ROUNDS

Figure 16

, *A—H*, each including three mutant alleles in the *R*-locus series as designated (Column 1). Each allele is mutant in one or more factors as shown in formulae (Column 2). Wild-type alternatives to mutant factors are designated +. The *O* subseries of alternative eye-color factors, given first in formulae, includes mahogany, *mh;* dahlia, *da;* orange, *or;* peach, *pe;* and oyster, *oy*. The *S* subseries, given second in formulae, includes dahlia, *da;* vermilion, *vm;* and scarlet, *st*. These are followed by recessive lethal, *l;* male-sterile, *ms;* female-sterile, *fsa;* or crossover-modifying, *C*, factors. Mutant factor *C*, perhaps an inversion, associated with mutant gene mahogany-819, reduces crossing-over between the *R* series and purple body color, *pu*, from 11 percent characteristic of other *R* alleles to one percent.

Formulae of genotypes are shown (Columns 3-8), alternating in successive generations. Under each genotype is given the eye color. In Merry-Go-Rounds, *C*, *E*, *F*, and *G* are shown in parentheses, the eye colors resulting from combinations with the independently segregating gene black, *bk*.

Merry-Go-Round B is exactly similar to A except that here a gene causing male-sterility, *oy*-816, replaces the lethal. In addition to the alternating males, oyster males therefore appear in each generation.

Of the three genes in Merry-Go-Round C, orange-336.2 is doubly recessive for color factors. Vermilion-809 is lethal at eclosion. Some of the vermilion-809 males are able to eclose and externally appear normal in structure but cannot walk, feed or mate. Their eyes are intense red with small masses of black pigment giving a dark appearance. This Merry-Go-Round has been combined with the gene black, *bk*, a recessive segregating independently of the *R* series. The different phenotypes produced by combinations with *bk* are given in parentheses. Black has the effect of removing all red or other bright pigment from the eyes changing wild-type brown and very dark reds to black. Lighter red eyes, eyes with reduced amounts of black, are changed to lavender and eyes lacking black such as most scarlets and tomato, orange and peach become oyster-white.

The gene mahogany-819 is not separable by inspection from wild-type brown, either in the males or in the homozygous females although the color may be slightly redder than the average of wild type. It is only in compounds with *O*-factor light color genes that the mahog-

any effect is produced and here it is distinct—a dark red easily separable especially in full lateral view. Mahogany-819 has the effect of reducing crossing-over with purple body color, *pu*, from 11 percent characteristic of other *R* alleles to approximately one percent.

Among the alleles of Merry-Go-Rounds D and E, dahlia-817 is complementary with dahlia-GF giving wild-type brown compound females. Oyster-805 is doubly recessive for color being a simple recessive to scarlet-DR. Because of the lethal factor in oyster-805, males do not develop and homozygous females cannot be obtained. That the eye would be oyster, is evidenced by the facts that compounds with viable oysters, like oyster-DR, are oyster and that the compound with peach-333.5 is tinged.

Merry-Go-Rounds F, G and H each include scarlet-474 as the lethal gene. In combinations with black, *bk*, in the even generations of F, the two genotypes of females are not separable by inspection. Individual settings must be made and fraternities including oyster males discarded.

In Merry-Go-Round G all males and half the females are oyster. Brown and scarlet alternate in the females to be retained. In combination with *bk* all females of the even generations are oyster. These must be set singly and the stock continued from the black daughters which are produced by half of these oyster females.[2]

In Merry-Go-Round H all females are scarlet with genotypes not separable by inspection. Because the female-sterile factor, *fsa*, is similar in the two non-lethal genes, the compound females are sterile.[3] Eye color of the female-sterile males, which are highly fertile, alternates oyster and scarlet in successive generations. The stock may be maintained in mass culture provided the different generations are not allowed to overlap. If females are set singly the half having the lethal gene are fertile. Females homozygous for *fsa*, whether compounds of genes *st*-689 and *oy*-423 or homozygous for either gene, sting and inactivate the host pupae, feeding on them and living for many days. Rarely they lay an egg which will hatch and develop into an adult.

The Merry-Go-Rounds of Figure 16 are selected as examples of the many that may be run with the large number of *R* alleles available. If apricot-837 (*ap.+.+*) were substituted for peach-333.5 in A and B the females to be discarded would be wild-type brown instead of scarlet. Thus they would be indistinguishable in the even generations from those to be retained and it would be necessary to make individual settings. If this substitution were made in D the females to be discarded would be pale apricot intead of tinged and the females to be retained would be brown in the odd as well as in the even generations. Thus eye color would alternate only in the males—oyster and apricot.

Substitution of oyster-801 (*oy.+.1*) for scarlet-474 in Merry-Go-Round H would make the scarlet sterile females separable by inspection from their sisters bearing the lethal factor. The latter would be brown in the odd generations and oyster in the even.

For greater variety of colors in some of the Merry-Go-Rounds the females may be kept heterozygous for black, *bk*, by mating to males with the gene *bk*. Thus as a starter in Merry-Go-Round C brown-eyed females may be isolated in an even generation, Generation 2, and mated to "oyster" males, stock *or*-336.2 *bk*. In Generation 4 the males will then be of four colors—orange, "oyster," vermilion and dark lavender. Matings of the brown females of Generation 4 to their "oyster" brothers or to stock *or*-336.2 *bk* males are then likely to bring out black and lavender in both sexes of Generation 5 as well as the colors previously expected. When black males, *mh*-819 *bk*, are obtained and these are crossed to their scarlet sisters, *vm*-809/*or*-336.2 +/*bk*, there may be expected in Generation 6, three colors in females—mahogany, brown and black, and four colors in males—orange, "oyster," vermilion and dark lavender.

The task of selection of males and females desired for mating is much light-

ened if it is carried out with pupae rather than with adults. Thus etherizing is unnecessary.

Discussion

Each member of the *R* series of mutant alleles causes impairment of one or more processes of the normal wild type. Impairment of any one process may be greater or less according to the action of the factor involved. Differences affecting a given process are due to homologous factors alternative to each other. These alternatives constitute different subseries within the *R* series of allelic genes. Theoretically the number of possible *R* alleles is equal to the product of the numbers of alternatives in the different subseries of factors. Genes having new combinations of alternatives are not obtained by crossing-over for the *R* alleles are apparently not pseudoalleles—hemialleles. The various combinations available have occurred by independent mutation.

Of the two subseries affecting eye color the *O* alternatives tend to remove or to inactivate some fundamental pigment-forming substance —"enzyme *O*." The most frequent mutation in *O* causes a completely pigmentless or transparent eye, oyster-white, with appearance somewhat darkened by the underlying black integument. Much less frequent mutations in *O* result in reddish, mahogany, dahlia, tomato, orange, peach and tinged eyes indicating amounts of pigment decreasing from the wild-type dark brown. The *S* alternatives decrease some secondary substance, "enzyme *S*," which in combination with enzyme *O*, produces dark (black) pigment. The most frequent mutation in *S* causes a brilliant red, scarlet, eye having very little or no black pigment. It is supposed that, in the production of scarlet, enzyme *O* acts alone having no enzyme *S* with which to unite. Mutations causing partial loss of enzyme *S* are roughly grouped as producing reddish, mahogany, dahlia and vermilion eyes. Actually an indefinite number of color grades occur both in the *O* and in the *S* subseries. Genes recessive in *O* are complementary with genes recessive in *S* giving wild-type compound females. Simple dominance or blending occurs between different members of the same subseries. Genes mutant in both subseries give mutant types in compound with genes mutant in either subseries. Males are always haploid and in eye color resemble homozygous females, except that in wild type and in some of the darker mutant types the males are somewhat lighter.

For convenience the subseries are placed arbitrarily in order *O.S.* Wild type, *+.+*, has an adequate amount both of enzyme *O* and of enzyme *S* to form dark brown eyes. Oyster, *oy.+*, has "no" enzyme *O* and scarlet, *+.st*, has "no" enzyme *S*. The compound females, *oy.+/+.st*, with dark brown eyes presumably have the normal amount of both.

O and *S* have been called "elements"[1] suggesting that the two processes might be carried out independently in different regions Because there is no evidence for this it may be preferable to designate them merely as factors determining certain processes. We know nothing of the minute structure or mechanisms involved.

R-allele mutant eye colors may be grouped as *shades, scarlets, tints* and *oysters*. The shades are red but have a considerable amount of black pigment. Thus far the genes for *shades* have proved to be mutant either in *O* or in *S*, not in both. They are called reddish, *rdh.+* and *+.rdh*, mahogany, *mh.+* and *+.mh*, dahlia, *da.+* and *+.da*, and vermilion, *+.vm*.

The scarlets are brilliant red with a very small black fleck or none. They are all scarlet, *st*, in *S*. In *O* they range from wild-type to scarlet as shown in tests with "complementary" oyster, *oy.+*. Thus *+.st* is completely complementary; *rdh.st, mh.st, da.st*, are partially complementary and *st.st* is a simple dominant over any *oyster* even of the complementary type, *oy.+*. The compound females showing partial complementarity illustrate different *shades* of eye color.

Until recently the *tints* obtained have all been scarlet in *S*. They are grouped as tomato, *to.st*, orange, *or.st*, peach, *pe.st*, and tinged, *ti.st*, in decreasing order of pigmentation. Actually tomato appears somewhat darker than scarlet. This is due to reduction in red pigment permitting incident light to penetrate the eye and to be absorbed in the black underlying integument. If dead dry specimens are compared, the scarlet eye remains brilliant red while the tomato tends tc be blotched with pigmentless areas.

An X-ray mutant type recently found following treatment of dahlia-GF, *da.+*, males has rather light but dull red eye color. Designated apricot-837 it has proved to be completely complementary with genes mutant in *S*. This is the first *tint* obtained representing an impairment of function *O* alone,—*ap.+*.

True or typical *oysters* have no pigment in their transparent eye. Thus far there have been obtained *oy.+*, *oy.rdh*, *oy.mh*, *oy.vm* and *oy.st*. Eye types called oyster but somewhat atypical are extremely light tinged, *ti.st*, and combinations of the *scarlets* and of the *tints* with black, *bk*. Black removes all red pigment but leaves some white. For this reason *or-336.2 bk* is a little whiter than typical oyster and *st*-DR *bk* is somewhat more opaque whitish.

Some of the *R* alleles cause malfunctions resulting in partial or complete sterility of males or of females. For example the compound females scarlet-689/oyster-423, *+.st.fsa. +/oy.st.fsa.+*, are sterile like the homozygotes. It may therefore be concluded that the function affected by the two genes is the same.

fsa. However, scarlet-841/oyster-423, *+.st. +. fsb/oy.st.fsa.+*, compounds are fertile so that these two genes are complementary with respect to fertility, the two malfunctions, *fsa* and *fsb*, being different and recessive. These fertile compound females, unmated, produce an abundance of (female-sterile) fertile males, scarlet and oyster in equal numbers.

Many of the *R* alleles have recessive lethal factors with characteristic effects. Some are egg lethals but others have their incidence in the larval or pupal stage. Vermilion-809 is called lethal at eclosion because the males pass through the pupal stage and many are able to eclose. Were they able to walk about and feed the factor might be characterized as male-sterile rather than lethal. Some truly male-steriles have been observed to mate but no daughters result. Homology of lethal and male-sterile factors cannot be determined. Because they cannot be transmitted by the male, only one can be brought into a cross.

It seems highly probable that many different processes concerned with viability and with male fertility as also with female fertility are performed by the germ plasm in such a way as to involve the *R* region. Thus *R*-locus mutations may interfere resulting in the malfunctions associated with this series. The hypothesis of the mere presence of separate gene elements to explain the normal processes of wild type alternatives to whatever we may find going wrong with mutant types appear inadequate. There are many different ways in which the germ plasm, a very complicated mechanism, may be disarranged.

Summary

An indefinitely great number of allelic genes occurs by mutation in a single series, *R*. These genes are pleiotropic bearing several factors of which two at least involve eye color. Mutations affecting a given factor form subseries of alternatives within the allelic series of genes.

The various new combinations of these alternatives are obtained, not by recombination between previously existing pairs of genes, but by mutation. There is, therefore, no evidence for pseudoallelism. All mutations are recessive to wild type and cause impairment or diminution of some normal process.

Of the two eye-color series, *O* determines the production of some primary substance, "enzyme *O*," essential to any eye pigment. Its mutations therefore range from near wild type brown to completely colorless, oyster white. *S* determines some secondary substance, "enzyme *S*," which uniting with enzyme *O*, gives rise to dark pigment. Its mutations therefore range from brown to scarlet.

A high proportion of the mutant genes cause sterility of males or of females or have a lethal effect. Factors essential to normal functioning are therefore affected.

Complementary allelism results in normal wild-type females compound for genes mutant in different factors.

A "Merry-Go-Round" is set up by combining three alleles including one lethal in an inbred pedigree. Females compound for the lethal are selected after sibling matings to produce the next generation. Genotypes of males and of half the females alternate in successive generations. An alternation of generations may therefore be observed in eye color.

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◆

CORN AND CORN IMPROVEMENT

THIS volume* is a compilation of papers prepared by different authors. The success of a book of this kind depends to a great extent upon the personnel selected to write the different chapters. In the opinion of the reviewer an excellent job of selection was done in obtaining the various authors to prepare the different sections. The men selected were all eminent in their fields. An idea of the comprehensive nature of this volume can be obtained from the chapter headings, also the list of authors who wrote the various parts. A list of these is presented herewith.

Chapter I. History and Origin of Corn, by Paul Weatherwax and L. F. Randolph. Chapter II. Vegetative Morphology, by John E. Sass. Chapter III. Structure and Development of Reproductive Organs, by Paul Weatherwax. Chapter IV. The Cytogenetics of Maize, by M. M. Rhodes. I think perhaps the readers of the *Journal of Heredity* will be as much interested in this particular

Corn and Corn Improvement, edited by George F. Sprague. Academic Press, Inc., New York, N. Y. $11.50. 1955.

chapter as any of the chapters in the book. There are about 100 pages in this section including a comprehensive bibliography. This is an up-to-date and thorough treatise on the cytogenetics of maize. The topics covered by Rhoades in this section include introduction, chromosome morphology, polyploidy, B chromosomes, meiosis in maize, crossing-over in maize, translocations, inversions, deficiencies, ring chromosome, abnormal chromosome 10 and mutations.

There is a comprehensive discussion of the work of McClintock's *Ac-Ds* system, also the similar findings of Brink in the modulator system (*Mp*) affecting the variegated pericarp in maize. There is a good discussion of the work of Stadler on induced mutations. Stadler's experiments were more extensive than those of any other investigator and he came to the conclusion that the X-ray induced mutants in maize were qualitatively different from the spontaneous ones. However, Stadler did find that some mutations induced by ultraviolet radiation seemed indistinguishable from spontaneous ones.

There is a good discussion of the compound *R* and *A* loci which were at one time thought to be multiple alleles, but it has now been clearly demonstrated that these are compound genes with the units very close together so that crossing-over rarely occurs. The work of Laughnan on the *A* series and the work of Stadler and his associates on the *R* locus have demonstrated conclusively that these loci are really not multiple alleles but are closely linked genes. They are separable by crossing-over, in some instances unequal crossing-over.

Chapter V is on Breeding, by G. F. Sprague. The readers of the *Journal of Heredity* will undoubtedly be interested in this treatise which includes after the introduction such things as mass selection, varietal hybridization, ear to row selection, selection in and among inbred lines, evaluation of inbred lines, the prediction of double cross performance, multiple hybrids and synthetics, recurrent selection, types of genic action, cytoplasmic sterility, insect resistance, disease resistance, and chemical composition.

Chapter VI is one on Mineral Nutrition of Corn, by J. D. Sayre. Chapter VII on Climatic Requirements, by Robert H. Shaw. Chapter VIII on Corn Culture, by G. H. Stringfield. Chapter IX is on Production of Hybrid Corn Seed, by John M. Airy; Chapter X on Popcorn, by Arthur M. Brunson; Chapter XI, Sweet Corn, by Glenn M. Smith; and Chapter XII on the Diseases of Corn, by Arnold J. Ulstrup, who is recognized as the leader in this field. Chapter XIII on the Most Important Corn Insects, by F. F. Dicke; Chapter XIV, Industrial Utilization, by G. F. Sprague; Chapter XV on the Nutritive Value of Corn, by Burke H. Schneider; and Chapter XVI, the final chapter, on World Production of Corn, by G. F. Sprague.

It is obviously impossible in a short review to do justice to any or all of the various chapters in this book on Corn and Corn Improvement. Its comprehensive nature makes it one which anyone working with corn will want to own.

In rereading the review which I have written I begin to wonder if I am not like the critic described by Oliver Wendell Holmes in that delightful little book "The Autocrat of the Breakfast Table." Holmes says, "As a general thing, I would not give a great deal for the fair words of a critic, if he is himself an author, over fifty years of age. At thirty we are all trying to cut our names in big letters upon the walls of this tenement of life; twenty years later we have carved it, or shut up our jackknives. Then we are ready to help others, and care less to hinder any, because nobody's elbows are in our way."

W. Ralph Singleton
University of Virginia
Charlottesville

RECESSIVE INHERITANCE OF SUSCEPTIBILITY TO POLIOMYELITIS

In Fifty Pedigrees

JOHN J. REEDY*

AS early as 1898, Taylor[6] presented evidence of a familial tendency toward poliomyelitis. Aycock[2] thought that there might be a genetic explanation for susceptibility to this disease, but he did not suggest any mode of inheritance. Addair and Snyder[1], after studying one extensive pedigree, proposed one autosomal recessive gene as the basis for susceptibility. Herndon and Jennings[4] studied cases which had occurred in 47 pairs of twins; their data also suggested an autosomal recessive gene with a penetrance of 35 percent.

The present study is based upon 50 pedigrees embracing over 5,600 individuals. It provides added confirmation for the hypothesis that susceptibility to poliomyelitis is conditioned by one pair of autosomal recessive genes. The calculated penetrance in the present study is over 80 percent.

The poliomyelitis vaccine presently in nation-wide use is intended to produce artificial immunity to paralytic poliomyelitis and may well mask the expression of the hypothesized gene in future generations.

Materials and Methods

In this study only paralytic poliomyelitis, in which the virus invades the central nervous system, was considered. The more common generalized infection, with no invasion of the central nervous system, was disregarded.

Through the cooperation of the Northern Indiana Crippled Children's Hospital and the South Bend office of the National Foundation for Infantile Paralysis, names and addresses were obtained of the 142 children admitted to Northern Indiana Crippled Children's Hospital for paralytic poliomyelitis during the epidemic of 1950.

Pedigrees were collected for 50 of the 142 patients. These were selected partly at random and partly on the basis of willingness of the families to cooperate and of their proximity to South Bend (most lived within 65 miles and all within 100 miles of South Bend). The 50 pedigrees embrace 5,677 individuals (2,712 males, 2,648 females, and 317 of unknown sex), including 153 cases of paralytic poliomyelitis (115 positive and 38 suspected).

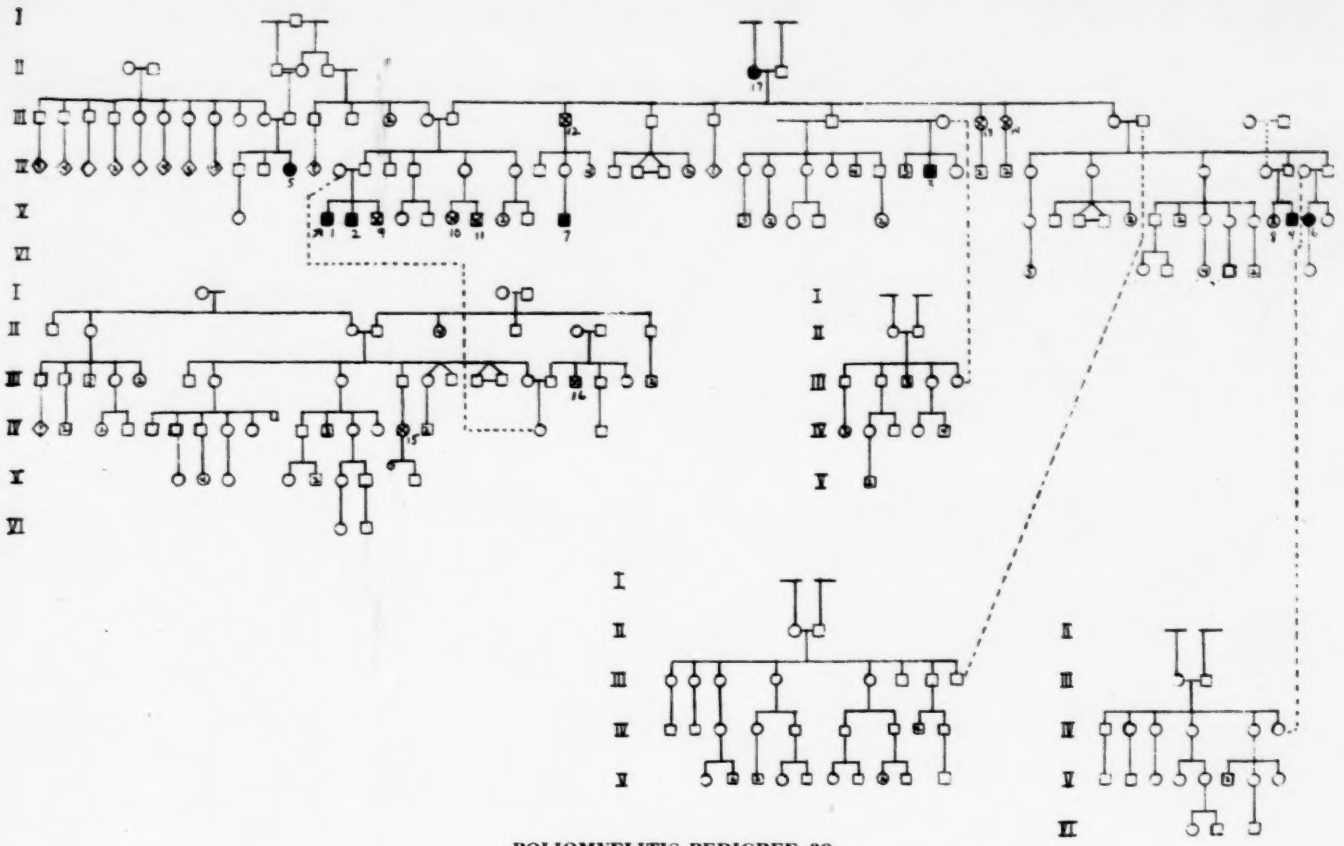
For each propositus, diagnosis was based upon examination of spinal fluid (141 by the South Bend Medical Foundation, and one at Indianapolis) and the judgment of the attending physician.

Cases of paralysis among relatives of propositi were found by interviewing the families. Diagnoses were confirmed and classified either as positive or suspected poliomyelitis by the following means:

(a) The attending physician, whenever possible, was contacted for verification of his reported diagnosis.

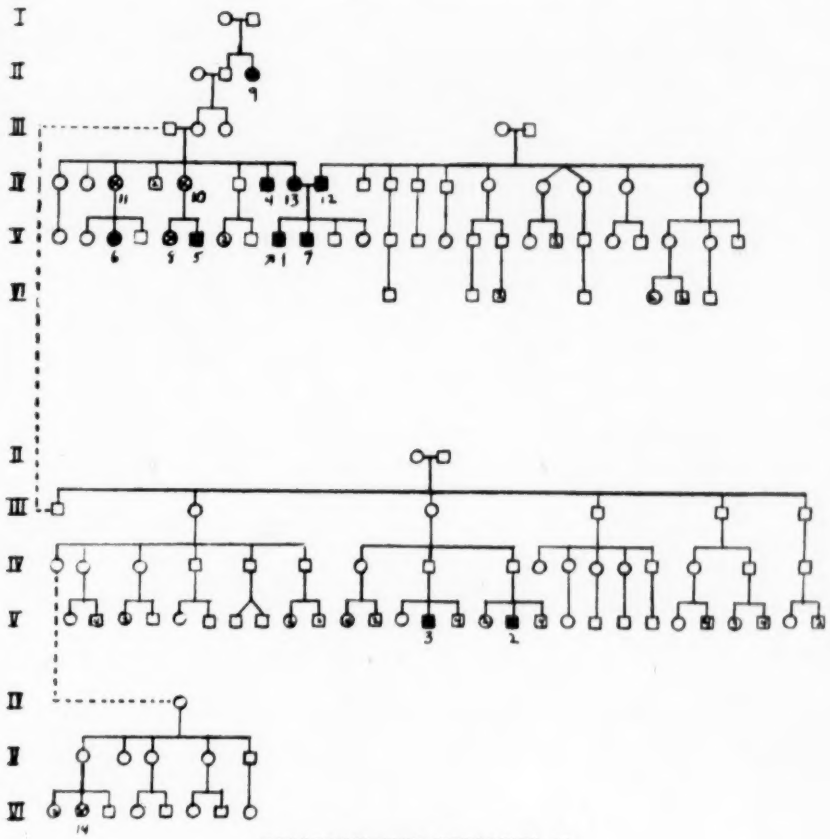
(b) When the attending physician

* University of Notre Dame. Present address: Stonehill College, North Easton, Massachusetts. This paper is part of a dissertation submitted to the Graduate School of the University of Notre Dame in partial fulfillment of the requirements for the degree of Doctor of Philosophy. The writer wishes to express appreciation and gratitude to Edward O. Dodson, Ph.D., University of Notre Dame, who directed this research; to Mr. Alvan A. Sauer, Superintendent of Northern Indiana Crippled Children's Hospital, for his generous cooperation and encouragement; to Leslie M. Bodnar, M.D., orthopedic surgeon, for diagnostic services and counsel; to C. Nash Herndon, M.D., Bowman Gray School of Medicine, for advice and encouragement; and to the families studied. Their cooperation and assistance made this paper possible.



POLIOMYELITIS PEDIGREE 39
Figure 17

The three pedigrees, 39, 40 and 46, published here, are representative of the 50 pedigrees studied. The numbers within squares, circles or diamonds indicate the total number of individuals observed. (See Pedigrees 40 and 46 for further description of symbols.)



POLIOMYELITIS PEDIGREE 40

Figure 18

This pedigree represents another sample of the 50 which were used as part of this investigation. The X's within squares and circles in this, and the other two pedigrees indicate suspected poliomyelitis.

was not available, the word of relatives was accepted if they were certain what the attending physician's diagnosis had been.

(c) When the diagnosis of the attending physician was not available, a full description of the case was submitted to an orthopedic surgeon for his judgment.

(d) All other cases were recorded as suspected rather than positive. Certain other diseases, such as meningitis and cerebral palsy, are sometimes confused with poliomyelitis. In this study, cases were always listed as suspected if there seemed to be any probability of such confusion.

The frequency of poliomyelitis among the relatives of propositi was compared with that of the northern Indiana population from which the sample was drawn.

The familial background of each sibship containing cases of poliomyelitis were investigated and secondary cases within a coefficient of relationship of $\frac{1}{8}$

were recorded.

Each pedigree was analyzed for evidence of conformity or non-conformity with the hypothesis of one autosomal recessive gene. Sibship analysis was used to test for conformity with expected results from the type of mating presumed to have occurred according to the hypothesis under consideration.

Finally, sex-linkage, Y-linkage, and sex-influenced patterns of inheritance were eliminated as possible explanations for the data.

Results and Discussion

A summary of the 50 pedigrees is presented in Table I. Since the propositi were the only positive cases in pedigrees 1 through 24, these have been summated for brevity.

Each pedigree was individually checked against the hypothesis and all were found to agree as exemplified by Figures 17, 18, and 19.

The sibship data are summarized in Table III.

Pedigrees 39, 40, and 46 are included here as Figures 17, 18 and 19 to illustrate extensive pedigrees containing numerous cases of positive and suspected paralytic poliomyelitis.*

The sample of 5,677 persons included 103 cases of poliomyelitis which occurred within the previous five-year period. If the 50 propositi and the 15 uncertain cases be eliminated to avoid bias, there remain 38 cases. This gives a rate of 6.75 per thousand. In contrast to this, the rate in the population of the area sampled was less than two per thousand in the same five-year period. That all cases would be reported to the National Foundation for Infantile Paralysis seems most likely, as financial assistance was available to them. This highly significant difference could be explained by genetic factors or by greater opportunity for transmission within families. That the latter is improbable is indicated by the fact that other cases in the families were often widely separated in space or time, while bed mates of propositi often escaped.

The number of sibships containing paralytic poliomyelitis falling within the various coefficients of relationship were determined for two groups of sibships, i.e. 100 sibships of positive paralytic poliomyelitis and 120 sibships including suspected paralytic poliomyelitis as well (Table II).

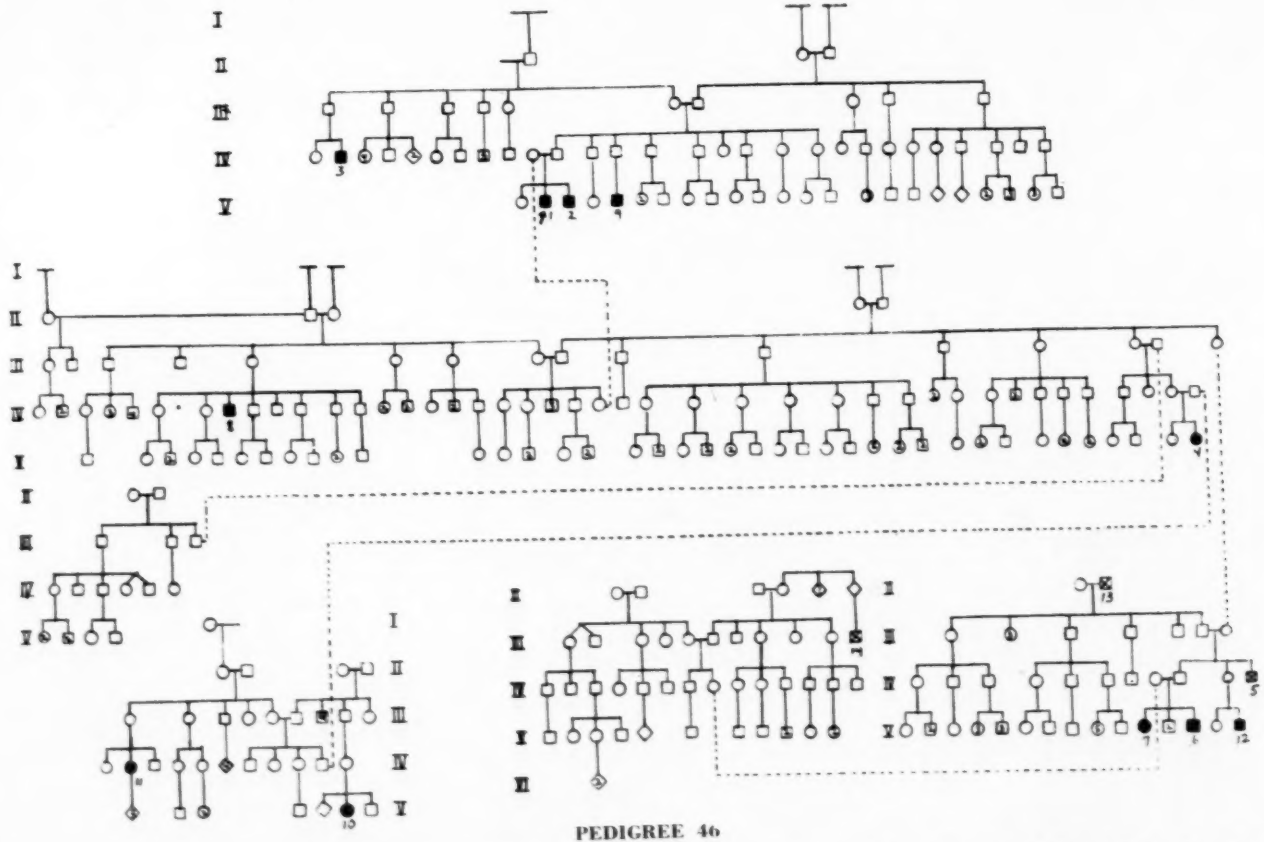
The majority of the cases were found in sibships of which both parents were normal. Using the correction of Bernstein[8] as found in Hogben[6] to eliminate the bias introduced by selection of pedigrees through propositi,

*All of the pedigrees and other data of this study are included *in toto* in the writer's doctoral dissertation, which is on file in the library of the University of Notre Dame.

TABLE I. Pedigree data

PEDIGREE NUMBER	Total No. ♂	Total No. ♀	Total No.‡	Positive ♂	Positive ♀	Suspected ♂	Suspected ♀
1 – 24	896	925	44	15	9	2	5
25	43	29	5	1	1	0	0
26	53	71	18	0	2	1	0
27	44	58	0	0	1	0	2
28	38	28	0	1	0	1	0
29	86	73	6	2	0	0	0
30	33	37	0	0	1	2	0
31	20	17	0	2	0	3	1
32	21	23	24	1	1	0	0
33	18	16	1	0	2	0	0
34	41	50	1	0	2	0	0
35	50	46	28	4	0	0	0
36	45	49	4	0	2	1	0
37	149	138	18	3	3	0	0
38	93	127	10	1	2	0	0
39	145	132	42	5	3	4	5
40	87	70	0	7	3	0	4
41	70	77	7	2	0	2	0
42	126	89	19	1	2	0	0
43	44	36	25	1	2	0	0
44	38	34	1	2	1	0	0
45	53	42	0	2	2	0	0
46	176	162	17	7	4	3	0
47	84	81	2	2	1	1	0
48	73	52	22	5	2	0	1
49	69	72	23	2	1	0	0
50	117	114	0	0	2	0	0
Total	2,712	2,648	317	66	49	20	18
				115		38	
Total Number of Individuals Studied—5,677				Total Number of Cases—153			

‡Sex Unknown.



PEDIGREE 46
Figure 19

The numbers below squares or circles in the three pedigrees correspond to code numbers used in the study. Dotted lines indicate the same person appearing in two places in the pedigree.

the number of cases expected in each sibship was determined. In Table III, Section A, in which only positive cases were considered, 135.75 cases were expected in 98 sibships. Actually 110 were observed. Dividing the observed by the expected number of cases leads to a penetrance of 81.0 percent. If suspected cases as well as positive cases be accepted (Section B) then the expected value in 112 sibships is 157.04. The observed number is 137, and the penetrance is 87.2 percent.

Some of the sibships were derived from parents one of whom was normal and one affected. Only one such sibship involved positive poliomyelitis exclusively (Section C). One case was expected and observed, hence the penetrance is 100 percent. If suspected cases be included (Section D), then there were seven such sibships, with 11.5 cases expected and 11 observed for a penetrance is 95.7 percent.

In only one instance (Table III, Section E, from pedigree No. 40, Figure 18) was a family found in which both parents had poliomyelitis. In accordance with the hypothesis, all of their children should be affected. Of the four children in this sibship, the oldest had had poliomyelitis when he was an only child; the second, the propositus, was promptly hospitalized when his case was discovered; the third child was then three years of age and had not yet developed poliomyelitis at the termination of this study; the fourth child was not born until long after the recovery of the propositus. The 50 percent penetrance in this sibship, therefore, cannot be considered inconsistent with the hypothesis that susceptibility depends upon a pair of autosomal recessive genes.

Collectively, the sibship data indicate a penetrance of 80.3 percent for positive cases only, and 88.2 percent for positive and suspected cases. These figures depend upon the number of homozygous recessive individuals and the number of clinical cases of poliomyelitis in the sample. Only the latter was observed, while the former was estimated by Bernstein's method. While the true value might be somewhat higher or lower than that calculated, it is clearly widely discordant with the penetrance of 35 percent reported by Herndon and Jennings.[4] This probably results from a difference in diagnostic criteria. Environment is an important factor in the clinical expression of paralytic poliomyelitis. The presence of the virus, how it is contacted, the physical condition of the individual contacting it (such as being excessively fatigued, chilled, or having recently undergone nose and throat operations), and many other environmental conditions may alter the clinical expression of paralytic poliomyelitis. Also, different strains of the virus, as well as their different virulence at different times may be a major factor in the penetrance of the hypothesized gene.

Sex-linkage, Y-linkage, and sex-influenced patterns of inheritance were all considered in relation to the data. These were eliminated not only because of the consistent concordance of the data with the hypothesis of a single autosomal recessive gene, but also because these patterns would require a significant deviation from the observed distribution of the cases between the sexes.

Among the 50 pedigrees studied there were 66 males and 49 females that had positive paralytic poliomyelitis. The probability that this represented a chance deviation from a 1:1 sex ratio was determined by the chi-square method. A χ^2 of 2.513, giving a P value of just under 0.120 was calculated. If the suspected cases are added to the above, 85 males and 67 females had paralytic poliomyelitis. The probability that this represented a chance deviation from a 1:1 ratio was also determined by the χ^2 method. A χ^2 of 2.359, giving a P value of approximately 0.133 was calculated. If a sex-linked or sex-influenced pattern of inheritance were applicable to the data a greater variance from a 1:1 sex ratio would be expected. A Y-linked pattern of inheritance is automatically ruled out by the occurrence of paralytic poliomyelitis among females.

An attempt was also made to distinguish genetically, between bulbar and spinal poliomyelitis, but no progress was made.

Summary

Pedigrees were compiled for 50 poliomyelitis patients. The pedigrees embrace 5,677 persons and include 153 cases of poliomyelitis, of which 115 were positive and 38 suspected.

The frequency of poliomyelitis among the relatives of the 50 propositi in a specified five-year period was calculated to be 6.75 per 1,000 while the corresponding rate in the gen-

TABLE II. Numbers of sibships falling within various coefficients of relationship

Coefficients of Relationship	Negative or Outside ⅛	Within ⅛	Within ¼	Within ½
100 Sibships Positive Paralytic Poliomyelitis	58	42	20	15
120 Sibships Including Suspected Paralytic Poliomyelitis	58	62	45	29

TABLE III. Sibship data

	No. of Children in a Family	No. of Sibships	Total No. of Children	Average No. Affected Children in a Sibship	Total No. Affected Children Expected*	Total No. Affected Children Observed
SECTION A. Positive Cases from Matings Normal × Normal ($Aa \times Aa$)	1	12	12	1.000	12.00	12
	2	29	58	1.143	33.15	33
	3	23	69	1.297	29.83	26
	4	13	52	1.463	19.02	16
	5	8	40	1.640	13.12	8
	6	4	24	1.825	7.30	5
	7	3	21	2.020	6.06	5
	8	2	16	2.222	4.44	2
	9	1	9	2.433	2.43	2
	10	2	20	2.649	5.30	2
	12	1	12	3.098	3.10	1
SECTION A Totals		98	333		135.75	110

Penetrance = $\frac{\text{Observed Cases}}{\text{Expected Cases}}$ = 81.0%

SECTION B. Positive or Suspected Cases from Normal × Affected ($Aa \times aa$)	1	18	18	1.000	18.00	18
	2	30	60	1.143	34.29	33
	3	23	69	1.297	29.83	28
	4	17	68	1.463	24.87	26
	5	8	40	1.640	13.12	8
	6	5	30	1.825	9.13	6
	7	3	21	2.020	6.06	5
	8	2	16	2.222	4.44	2
	9	2	18	2.433	4.87	6
	10	2	20	2.649	5.30	2
	12	1	12	3.098	3.10	1
	16	1	16	4.041	4.04	2
SECTION B Totals		112	391		157.04	137

Penetrance = 87.2%

SECTION C. Positive Cases from Normal × Affected ($Aa \times aa$)	2	1	2		1.0	1

Penetrance = 100%

SECTION D. Positive or Suspected Cases from Normal × Affected ($Aa \times aa$)	2	3	6		3.0	5
	3	3	9		4.5	3
	8	1	8		4.0	3
SECTION D Totals		7	23		11.5	11

Penetrance = 95.7%

SECTION E. Positive Cases from Affected × Affected ($aa \times aa$)	4	1	4		4.0	2

Penetrance = 50%

* The expected number of children was calculated by using the correction of Bernstein[8] as found in Hogben.[5]

eral population of that area was under two per thousand during the same period.

Over 40 percent of all sibships containing paralytic poliomyelitis showed a familial history of secondary cases occurring in sibships having a coefficient of relationship of ⅛.

Sibship analysis showed that all of the data were consistent with an hypothesis of a single autosomal recessive gene as the basis of susceptibility to poliomyelitis.

On the basis of the data of this study, the hypothesized gene for susceptibility to poliomyelitis has a penetrance on the order of 80 percent.

The difference in incidence of poliomyelitis among males and females is not statistically significant, hence sex-linkage and sex-influenced patterns of inheritance are ruled out. Y-linkage is ruled out by the common occurrence of the disease in females.

The data were not adequate to distinguish any genetic difference between bulbar and spinal types of poliomyelitis.

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